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THE Journal OF THE FLORIDA MEDICAL ASSOCIATION

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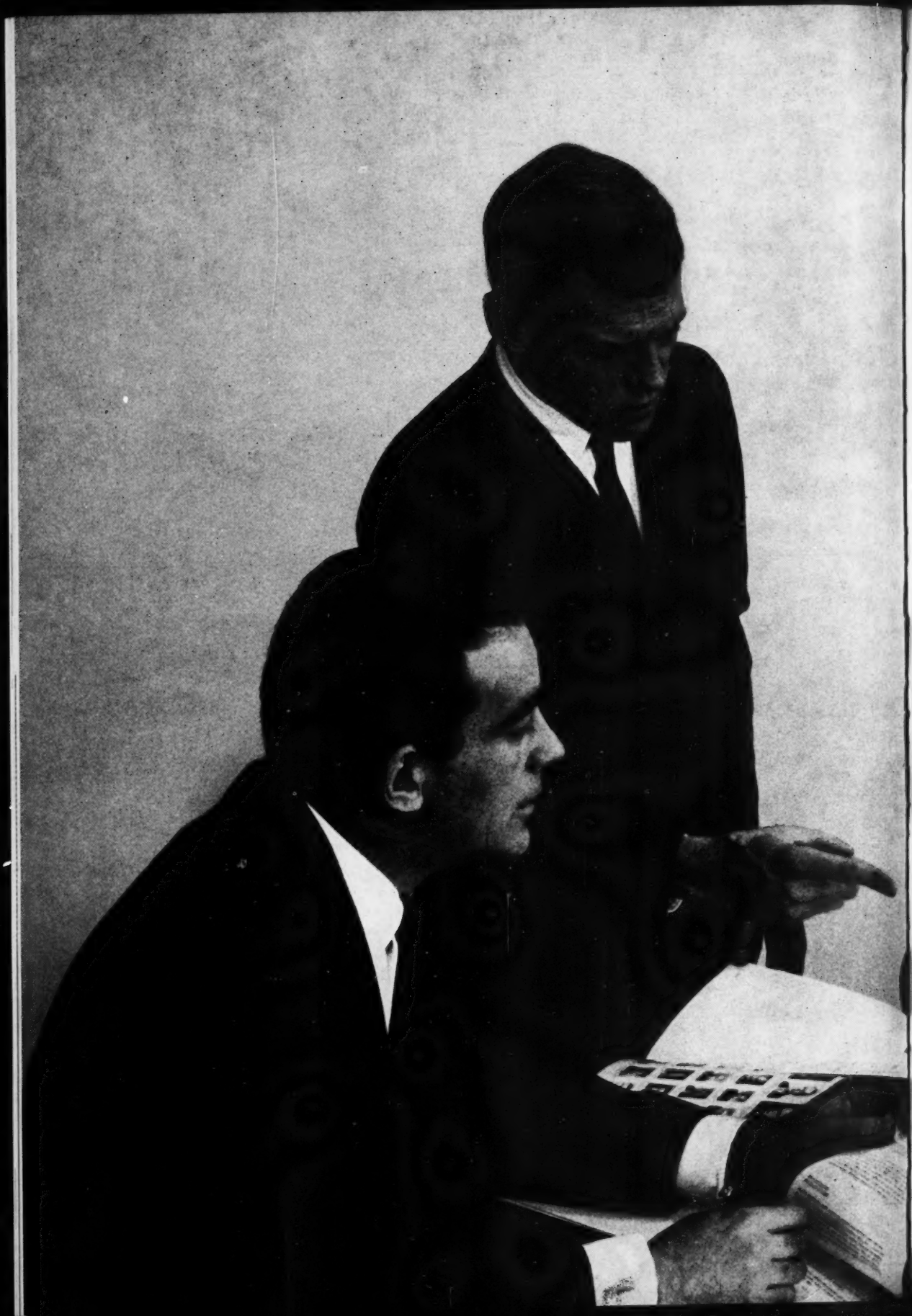

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Actual case summary from the files of Bristol Laboratories' Medical Department

The Last Word . . .

The Meaning and Challenge Of the 1960 Federal Welfare Law Amendments

H. PHILLIP HAMPTON, M.D.
TAMPA

The amendments to the federal welfare laws enacted by the 86th Congress provide more money beginning Oct. 1, 1960 to pay vendors of medical care of all types to needy sick 65 years of age and older.

In order to obtain these federal matching funds the individual states must provide 20 per cent to 50 per cent of the money spent (depending on the per capita wealth—Florida 25 per cent) and institute a medical care program equally applicable in all sections of the state.

For those receiving old age assistance (OAA approximately 70,000 in Florida) the federal government will participate (75 per cent in Florida) in establishing a pooled fund of up to $12 per month per recipient ($10,000,000 in Florida) out of which vendors of medical care to OAA recipients will be paid upon submission of approved vouchers to the state agency administering the fund. "Vendors of medical care" may include hospitals, nursing homes, drugs and appliances, outpatient and inpatient medical services, and visiting home medical care.

This part of the recent amendments is not new, but the amount of federal participating money earmarked for medical care (Kerr amendment) has been increased. Florida now has in operation a vendor payment medical care program for Public Assistance Recipients providing short term hospital care and drugs. The state law permits other medical services, but sufficient funds have not been available. The State of Florida is now appropriating into separate pooled funds to provide short term hospital care and drugs for PAR (approximately 75 per cent used for OAA recipients) at the rate of almost $700,000 this past quarter matched by $1,000,000 of federal funds. Beginning Oct. 1, 1960, if the quarterly contribution to this fund from within the state (not necessarily the state treasury) was increased by $250,000, then $750,000 of federal funds would be added providing an amount sufficient to pay (in addition to the present PAR hospital and drug program) for all medical, hospital, nursing home and outpatient care services to those 65 and over receiving old age assistance, now being provided by individual counties from funds derived 100 per cent through county taxes.

The other part of the amendment to the federal welfare laws by the 86th Congress (Mills amendment) provides federal matching monies to aid in giving medical care only to a new category in the federal Public Assistance program—those 65 years of age and over not eligible for OAA subsistence payments by reason of their personal resources or other reasons, but in need of financial help because of illness as determined by standards established within the state. Our experience in Florida with the Hospital Service for the Indigent program (in operation since 1955) which includes those 65 years of age and over in need of short term hospital care and unable to pay the cost, would indicate a population of about 50,000 people in Florida age 65 years and over eligible for public assistance medical care and not on OAA.

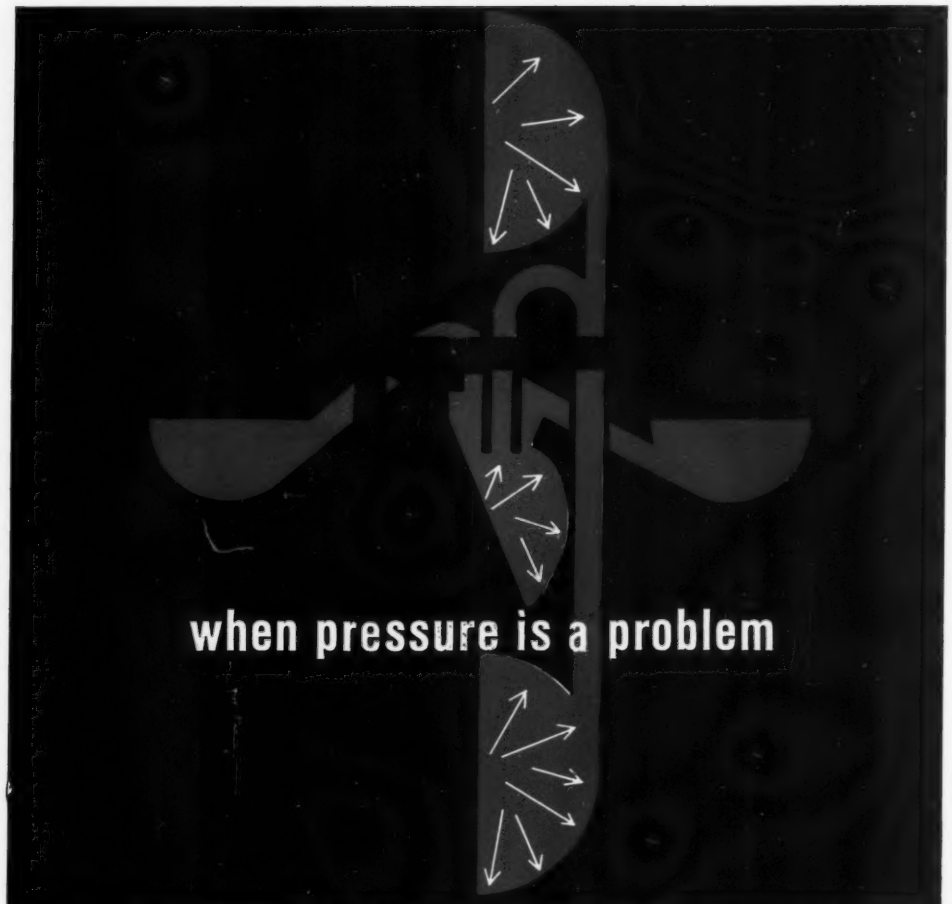
A comprehensive medical care program (hospital care, drugs, outpatient medical care and visiting nurse services) could be provided for all needy sick 65 years of age and over in this state, beginning any time after the first of October 1960, with the federal government paying 75 per cent of costs through existing state laws, if:

1. The county commissioners would agree that all counties appropriate one-half dollar per capita annually from presently budgeted welfare medical funds to match state funds of $1 per capita, the federal matching would provide a fund sufficient to pay all vendors of medical care to the aged needy sick, continuing the present hospital and drugs programs for public assistance recipients to which the state is now appropriating about $1 per capita and the hospital service for the indigent program to which 98 per cent of counties by population are now contributing 50 cents per capita.

2. The State Department of Public Welfare

(Continued on page 411)

Chairman, Council on Legislation and Public Agencies, Florida Medical Association.



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THE JOURNAL
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Volume XLVII, No. 4, October, 1960

Therapy of the Systemic Mycoses

JOHN J. PROCKNOW, M.D.
CHICAGO

The systemic or deep mycoses warrant an increasing prominence in the differential diagnosis of acute or chronic infection, particularly when pulmonary involvement is manifest. Intensified therapy with broad spectrum antibiotics, steroids, radiation and antineoplastic drugs, improvement in life expectancy and extensive travel have all served to increase the incidence of systemic fungal disease. The physician must have a high level of suspicion of a deep fungal infection to orient him to that most important matter of making an early diagnosis. It is this early diagnosis which insures effective treatment as a consequence of the immediate application of properly selected therapeutic measures and drugs.

The management and treatment of the deep mycoses are still plagued with ignorance, prejudice, nostrums, toxicities and ineffectiveness. What may be effective against one fungus infection may be completely ineffective against another. Multiple therapeutic agents have been advocated and most have been discarded as they fail to weather the mycotic storm.[1-3] Only a few drugs have proved worth while or specific and they will be briefly reviewed in relationship to those mycoses which they benefit. Conventional supportive measures should always accompany the administration of any specific therapeutic agent in the treatment of systemic infection with fungi. A basic understanding of the nature of the fungal agent and the type of lesion produced aid in the understanding of the reasons for therapeutic success or failure.

Associate Professor of Medicine, University of Chicago, School of Medicine.
From the Section of Preventive Medicine, Department of Medicine, University of Chicago, School of Medicine.
Read before the Florida Medical Association, Eighty-Sixth Annual Meeting, Jacksonville, April 9, 1960.

Amphotericin B (Fungizone-Squibb)[4] has proved the most promising of any of the antifungal agents discovered to date and, therefore, deserves special consideration. It is the first time that an antibiotic has been termed "broad spectrum" against the fungi. Amphotericin B is a polyene antibiotic produced by a streptomyces strain first isolated in 1955 from a soil sample obtained in Venezuela.[5] It is marketed as a lyophilized yellow powder made soluble by the addition of sodium desoxycholate. The drug is very poorly absorbed through the gastrointestinal tract so that the oral route of administration, as well as the subcutaneous or intramuscular routes, have proved quite ineffectual clinically and of limited value. Demonstrations of its broad fungicidal potency in vitro, its efficacy against experimental mycoses in animals and the increasing numbers of beneficial results being reported in human cases verify that treatment with amphotericin B is successful in histoplasmosis, coccidioidomycosis, cryptococcosis, North American blastomycosis and candidiasis. There has been some beneficial effect noted in the treatment of sporotrichosis, aspergillosis and mucormycosis. An effective serum concentration ranging from 0.5 to 2.0 μg./ml. of the drug against these pathogenic fungi can be obtained only by intravenous administration of the drug.

When definite indications for therapy have been established and the patient has been committed to its use, amphotericin B must be employed at full dosage and with persistence and ingenuity. The course of therapy is initiated with a dosage of 0.25 mg./kg. diluted in 500 to 1,000 ml. of 5 per cent glucose in water given over a six

hour period and increased by daily increments over a period of one week to an average daily dosage of 0.75 to 1.5 mg./kg. It is imperative that the dosage level of 1.5 mg./kg. per day is not exceeded. To avoid toxicity, the drug may be given on alternate days since an inhibitory concentration in the blood will be maintained for 48 hours. The duration of therapy has not been established, but may vary from many weeks to many months, depending upon the severity and kind of mycotic infection. Overtreatment with amphotericin B should be the rule to insure against relapse. Several factors limit this ideal approach, however. The route of administration, the unpleasant side effects and the need for prolonged therapy all jeopardize a patient's acceptance of the drug.

The impermeability of the meningeal barrier to amphotericin B offers another challenge to its effectiveness in the treatment of mycotic meningitis. In the disseminated mycoses having a predilection for the central nervous system, heroic measures are often necessary. A high serum level of amphotericin B acquired by intravenous infusion provides only a negligible level in the spinal fluid. Intrathecal administration of amphotericin B is, therefore, advocated concomitantly with intravenous therapy to provide inhibitory levels of the drug in the spinal fluid when meningeal involvement has occurred. The level of spinal fluid protein, however, bears some direct relationship with the amphotericin B level following intravenous administration.

The most common acute toxic reactions to amphotericin B are anorexia, nausea, vomiting, chills, fever, headache, malaise, diarrhea, tachycardia and apprehension.[7] They occur more prominently in the early course of intravenous therapy. Anxiety and gastrointestinal symptoms are suppressed by the administration of a sedative or tranquilizer and antiemetic agent. The chills, fever, headache and malaise may be checked by acetylsalicylic acid before and during therapy. Systemic reactions in general can be modified by the use of 10 to 25 mg. of hydrocortisone in each infusion or by antihistaminics given before administration. Any tissue irritation characterized by pain, inflammation or thrombosis at the injection site may be prevented by 20 mg. of heparin added to each infusion. Urinalysis may reveal albuminuria, casts and even hematuria. An elevation of blood urea nitrogen frequently occurs and necessitates discontinuance or lowering of the dosage of amphotericin B. Complete reversibility of the azotemia to normal occurs within a week of drug cessation. Suppression of renal physiology does not always necessarily require discontinuance of the drug because the blood urea nitrogen in many cases may not rise much above 50 mg./ml. in spite of continued therapy. Probable future improvements in this preparation may provide greater ease in administration and decreased toxicity. The saving grace in the imperfections of amphotericin B is the limitation they impose upon its indiscriminate use.

Although there is laboratory evidence that resistance to amphotericin B may develop,[7,8] clinical evidence is lacking to support this view.

Histoplasmosis

Through significant numbers of clinical trials, amphotericin B has proved to be the most efficacious agent in the treatment of histoplasmosis to date.[10-13] Histoplasma capsulatum is inhibited by the small amount of 0.4 μg. of the drug per milliliter of media,[6] and it is highly protective to animals infected experimentally.[14] Acute pulmonary histoplasmosis, a benign infection, frequently encountered in the midwestern United States, is self-limiting and usually warrants no treatment other than supportive measures. The specific indications for therapy in histoplasmosis include (a) severe primary infection, (b) persisting symptomatic infiltrations threatening to disseminate, particularly in infants, (c) mucocutaneous lesions, (d) disseminated infection, (e) chronic pulmonary infection with cavitation, (f) histoplasmosis superimposed upon chronic debilitating disease, and (g) surgical resection.

The most impressive use of amphotericin B has been in the suppression of infection in the disseminated and chronic cavitary forms of histoplasmosis. After a few weeks of treatment, positive sputum promptly converts to negative, accompanied by a marked decrease in the amount of sputum. Although significant symptomatic relief is afforded the majority of patients, roentgenographic evidence of improvement infrequently occurs in the chronic cavitary form of the disease. In cases of disseminated or acute pulmonary histoplasmosis, however, treatment results in rapid improvement in roentgenographic findings. The complement-fixing antibodies generally fall in titer as therapy is continued, but only occasionally reach negative titers.

There is a tendency in chronic cavitary cases of histoplasmosis for relapse to occur with return of symptoms and positive sputum. The chronic fibrosis and extensive necrosis characteristic of chronic cavitary disease prevent the drug from penetrating the lesion at sufficiently high levels to be fungicidal. Amphotericin B is primarily suppressive in this form of histoplasmosis. Consequently, the course of therapy must be as protracted as the patient and the drug permit. Even four to five month courses of intravenous amphotericin B have failed to prevent relapse. Surgical resection of the chronic lesion covered by intravenous amphotericin B offers a successful approach in many cases of cavitary histoplasmosis.

The only other antifungal agents in the treatment of histoplasmosis worthy of mention are the sulfonamides. There have been some doubts cast upon their effectiveness both in experimental and clinical trials. Christie,[15] however, has been successful in achieving remissions in cases of disseminated and mucocutaneous histoplasmosis. Sulfadiazine is preferable and given orally at 6 Gm. daily or in sufficient amount to maintain the blood level at 10 to 20 mg. per hundred milliliters for several months.

Coccidioidomycosis

Amphotericin B is absolutely the first therapeutic agent that has been effective in the treatment of coccidioidal disease.[16-18] It possesses a high degree of activity in vitro against Coccidioides immitis, inhibiting the organism with as little as 0.5 μg. of the drug per milliliter of media.[6] Primary coccidioidomycosis occurs very commonly in the endemic southwestern United States and usually requires no treatment. The true indications for amphotericin B treatment in coccidioidomycosis are (a) severe primary infection, (b) primary infection threatening to disseminate because of known susceptibility of the Negro, Filipino, Mexican and pregnant woman, (c) disseminated infection, (d) skin granulomas and abscesses, (e) negative reactors to the coccidioidin skin test, (f) unstable serology, (g) coverage for surgical procedures.

Since the complement fixation and precipitin tests are of the highest specificity in coccidioidomycosis, they serve as a valuable prognostic guide to therapy.[19,20] If serologic regression to amphotericin B is good, chances of relapse are minimal. If relapse does occur, repeated courses of therapy should be given. The intravenous mode of administration can be combined with intrathecal treatment in cases of coccidioidal meningitis.[18] Surgical resection of chronic cavitary lesions has resulted in significant curative rates with lowering of complement fixation titers. Surgical intervention combined with effective amphotericin B therapy is preferable in the treatment of chronic manifestations of the disease.

Cryptococcosis

It is suspected that many cases of benign cryptococcosis, acquired by the inhalation of dust of pigeon excreta infected with Cryptococcus neoformans, go undiagnosed. The primary lesion develops in the pulmonary tissue, but dissemination may involve all organ systems with a special predilection for the central nervous system. It is in the nervous tissue that the organism proliferates and becomes thickly encapsulated, thus increasing its resistance to the body defenses. Prognosis becomes extremely poor when the nervous system is involved. The earliest possible diagnosis of any cryptococcal infection leading to prompt therapy would provide the greatest chance of cure. Too often, the disease is recognized only after central nervous system involvement has occurred. Without therapy, cryptococcal meningitis will usually culminate in death within four to six months. Spontaneous remissions in this disease have greatly confused the efficacy of multiple therapeutic agents advocated in the past.

Amphotericin B, although admittedly not wholly the ideal therapeutic agent, has been the most promising agent in the treatment of cryptococcosis.[17,21-23] The minimal inhibitory concentration of amphotericin B at 0.03 μg./ml. of media for the cryptococcus fungus is the lowest for all pathogenic fungi.[6] Although the serum levels of amphotericin B effective against Cryptococcus neoformans can be easily attained intravenously, the blood-brain barrier blocks the antibiotic from achieving a concentration sufficiently high to deal effectively with the organism in the spinal fluid. An impressive number of patients, however, with cryptococcal meningitis treated only with intravenous amphotericin B have clinically improved. It may be concluded that even a small amount of antibiotic in the spinal fluid is capable of inhibiting some strains of Cryptococcus neoformans. Relapses frequently occur after prolonged intravenous amphotericin B therapy. Intrathecal injections of amphotericin B have been used successfully in reducing relapse

rates.[10] This method is not without hazards, but does insure a fungicidal level for cryptococcus within the spinal fluid for at least 24 hours after infection.[6] Surgical excision of early localizing lesions still plays an important role in removing any focus for possible dissemination to the central nervous system.

Blastomycosis

North American blastomycosis is a chronic granulomatous disease caused by the fungus, Blastomyces dermatitidis. The primary portal of entry is the respiratory tract, but dissemination may occur to involve other organs of the body, with predilection for skin, subcutaneous tissue, and bone. Blastomycosis can now be classified into (a) primary cutaneous inoculation blastomycosis, and (b) systemic blastomycosis. Both forms require treatment. The aromatic diamidines and amphotericin B are the drugs of choice.

The more stable and least toxic of the diamidine compounds, 2-hydroxystilbamidine, had gained widespread acceptance before the advent of amphotericin B in the treatment of blastomycosis. Numerous case reports attest to the success of this drug.[24-26] A daily intravenous dosage of 225 to 450 mg. is given by slow intravenous drip. The total course of therapy should be between 9 and 12 Gm. The drug may be given continuously or in courses alternating with rest periods. It is thought that 2-hydroxystilbamidine is curative for systemic blastomycosis when sufficient immunologic defenses have been demonstrated by the patient, indicated by the presence of a positive blastomycin skin test and negative complement-fixing antibody titer.[27] Relapses or therapeutic failures with the aromatic diamidines will occur with organisms found to be drug-resistant or in patients who do not demonstrate a favorable immune response.

Amphotericin B is now beginning to appear superior to the aromatic diamidines in the treatment of North American blastomycosis.[28] It is particularly gratifying that successful cures have been achieved in persons who have relapsed following massive therapy with the aromatic diamidines. It has also been noted that a smaller total dosage of amphotericin B is capable of curing blastomycosis than is needed to cure the other systemic mycoses. Furthermore, repeated administration of amphotericin B has not resulted in drug resistance. Surgical excision of localized or chronic blastomycotic lesions should be practiced in carefully selected cases. If pulmonary resection is undertaken for a cavitary lesion, therapy with amphotericin B should accompany the procedure.

Actinomycosis

Actinomycosis, a chronic suppurative mycotic infection, can be classified as (a) cervicofacial, (b) abdominal and (c) thoracic. The latter two forms are decidedly less amenable to treatment. Spread of the infection is by contiguous invasion, resulting in the formation of abscesses which drain through multiple sinus tracts. Extensive fibrosis about the lesions tends to seal off the infection and thus complicates the effectiveness of therapy. The fungus, Actinomyces bovis, is an anaerobe and manifests itself in tissue as a filamentous colony, densely clustered, and offering further resistance to penetration by chemotherapeutic agents.

Penicillin is the drug of choice in the treatment of actinomycosis.[29-31] The actinomyces organism is extremely sensitive to penicillin in vitro. Furthermore, clinical confirmation of the value of penicillin in the treatment of actinomycosis has been overwhelmingly favorable. Frequent treatment failures occurred until more intensive and prolonged periods of treatment were undertaken. When massive doses of penicillin are combined with surgical excision or drainage of infected tissues, notable improvement in the more serious abdominal and pulmonary forms of the disease will usually occur.[31] The dosage ranges from 1 to 6 million units of penicillin daily for several months. Depending upon the severity of the case, the daily administration of penicillin can be increased to 12 million or more units or the period of administration may be prolonged up to a year or longer.

At times, treatment failures occur because of inadequate therapy, resistance of the organism, or sensitivity of the patient to penicillin. Even discounting these factors, the use of huge doses of penicillin may not arrest the disease process. In vitro sensitivity studies provide the best means of selecting a supplemental or substitute agent for penicillin. Erythromycin is one of the most effective antibiotics next to penicillin. The broad spectrum antibiotics, namely, the tetracyclines and chloramphenicol, are also active drugs against the actinomyces and cures have been reported with their use alone or in combination with other agents. Streptomycin and the sulfonamides are the least effective. Iodides have almost

completely lost their claim to fame in the therapy of actinomycosis. The surgical drainage or excision of infected tissue plays a very important role in the success of antibiotic therapy.

Nocardiosis

Nocardiosis is a fungus infection which is closely akin to actinomycosis. The pulmonary tract also serves as the usual portal of entry, from whence hematogenous spread occurs with predilection for the brain and subcutaneous tissues. Although the infection is chronically suppurative, it is less likely that sinus tracts and abscesses will form. Like Actinomyces bovis, nocardia is a bacterial-like fungus, but grows aerobically on many laboratory media. The organism may easily be confused with Mycobacterium tuberculosis because it often segments into short fragments and is frequently acid-fast.

Infection with the nocardia organism is more difficult to treat than actinomycosis because of greater severity of the infection and greater resistance of the organism. Sulfadiazine is the drug of choice in the treatment of nocardiosis.[31,33] In many clinical trials, recovery in nocardiosis was achieved only when the sulfonamides were added to other forms of therapy. The dosage of sulfadiazine ranges from 4 to 8 Gm. or more daily, or an amount sufficient to maintain a blood level of 10 to 20 mg. per hundred milliliters depending upon the severity of the infection. Since nocardia is quite resistant to therapy, sulfadiazine combined with streptomycin or a broad spectrum antibiotic offers a better chance of cure. Antibiotic therapy must be vigorous and prolonged for several months to several years to insure healing.

The choice of any supplemental antibiotic is usually based upon sensitivity tests, but such tests against nocardia have proved to be misleading. The broad spectrum antibiotic is given in the dosage of 2 Gm. per day. Surgical procedures to insure adequate drainage of abscesses and excision of locally infected tissues must augment the chemotherapeutic approach to nocardiosis. Furthermore, treatment with sulfadiazine alone or combined with an antibiotic should accompany all operative procedures.

Candidiasis

The fungus, Candida albicans, is an ubiquitous saprophyte existing normally in man within the mouth, gastrointestinal tract and vagina and on the skin. It can cause superficial infections of these tissues as well as assume the role of a serious pathogen in its invasion of the gastrointestinal mucosa and the bronchopulmonary system and then ultimately disseminate. The predisposing factors to systemic candida infections are debilitating diseases, infancy, diabetes mellitus, pregnancy, poor nutrition, obesity and chronic pulmonary disease.

Both nystatin and amphotericin B are equally effective antibiotics against Candida albicans by in vivo and in vitro studies.[5,34] The poor absorption of nystatin from the gastrointestinal tract limits its usefulness in the treatment of pulmonary or disseminated forms of candidiasis. Since it is very effective upon direct contact with the candida organism, available topical and oral formulations will cure the localized oral, cutaneous and vaginal infections and suppress gastrointestinal infections. In combination with tetracyclines it has gained acceptance in the prevention of monilial superinfection which might arise from prolonged broad spectrum antibiosis.

The drug of choice in the treatment of pulmonary and systemic candidiasis is amphotericin B.[4] The lowest minimal inhibitory concentration of the agent for Candida albicans is 0.5 μg./ml. of media.[6] Oral amphotericin B is also effective in gastrointestinal candidiasis.[35,36] The supplementing of oral or local forms of amphotericin B and nystatin with parenteral amphotericin B will provide intensive therapy for the most serious candida infections.

Methylrosaniline chloride (Gentian violet) was successfully used topically and intravenously for candidiasis before the advent of nystatin or amphotericin B. It is mentioned only as an alternate therapy to the newer agents.

Aspergillosis

Most cases of aspergillosis are caused by Aspergillus fumigatus. As a primary or secondary invader of lung tissue, it is most frequently associated with predisposing factors such as debilitation or diabetes. Secondary infection with aspergillus is commonly superimposed upon an antecedent pulmonary disease.[37]

Since no specific drug therapy has been particularly effective, the treatment of choice in localized pulmonary aspergillosis is surgical resection or drainage of focal infection.[38] The careful clinical control of the predisposing diseases is also very important.

Of the antifungal antibiotics, nystatin and amphotericin B deserve mention. Amphotericin B, in particular, needs further evaluation. Although the inhibitory effect of this drug on the aspergillus organism is insignificant by in vitro studies, in vivo studies on rabbits showed suppression of lesions in the treated animals.[39] Some, but all too few, clinical trials with amphotericin B have produced encouraging results.[17] Effective therapy with nystatin has been produced when administered intrapleurally or by inhalation in a few cases.[40-42]

Sporotrichosis

Sporotrichosis is often a chronic fungal infection caused by Sporotrichum schenckii. Infection is usually limited to the skin and subcutaneous tissues, but dissemination may occur to involve many organs.

Iodides are specific in the treatment of sporotrichosis, particularly in the localized and lymphatic form of the disease.[43] The disseminated forms of sporotrichosis, however, have been less responsive to iodide therapy. Iodides should be tried first as treatment for any form of sporotrichosis, nevertheless, and for sufficiently long periods. Response is slow, but quite often satisfactory, to the oral administration of a saturated solution of potassium iodide. It is prescribed in the initial dose of 3 to 5 drops given three times daily and increased by daily increments of 1 to 3 drops per day up to a total of 40 to 50 drops three times daily. Clinical evidence of improvement will generally be noted within a few weeks to a few months, but iodide therapy should be continued for at least another month after apparent cure is attained.

In those patients with generalized sporotrichal involvement unresponsive to iodide therapy or who are sensitive to iodides, a course of therapy with either 2-hydroxystilbamidine or amphotericin B is indicated. Both the aromatic diamidines[44] and amphotericin B demonstrate good in vitro effectiveness against Sporotrichum schenckii. The yeast phase of the organism is inhibited by amphotericin B in the small amount of 0.07 μg./ml. of media.[6] Sufficient curative value in a few cases of systemic sporotrichosis has been shown with these two drugs to warrant a clinical trial when iodides fail.[6,45]

Mucormycosis

Mucormycosis is a rare fungal disease entity and is caused by fungi of the Mucor, Rhizopus, and Absidia genera. Being very widely distributed in nature, these organisms can gain entrance primarily into the respiratory tract. There is a true predilection for persons suffering from uncontrolled diabetes mellitus. The only measure advocated thus far has been the careful control of the diabetic state which predisposes to the disease. Amphotericin B again shows possibility as the therapeutic agent in the treatment for mucormycosis and should be explored further. Recent studies in experimentally infected rabbits have shown very significant protection against lethal doses of Rhizopus spores and suppression of disseminated lesions.[46,47]

Conclusion

The treatment of systemic mycotic infections has at last achieved a bright outlook. Amphotericin B must be credited with a spectacular record in the broad approach to specific antibiotic therapy against multiple fungal agents. We can continue to hope that modifications in this therapeutic agent will decrease its toxicity and that our present recognition of increasing numbers of systemic fungal infections will culminate in the discovery of newer and better agents.

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Swimming Pool Granuloma

JOSEPH FARRINGTON, M.D., NELSON A. MURRAY, M.D.,
AND
MILDRED JEFFRIES, B.S., M.P.H.*
JACKSONVILLE

Traditionally, the pool swimmer is susceptible to eye, ear, nose and throat infections. Such skin infections as ringworm, schistosomiasis and scabies, and also gastrointestinal disorders, have less frequently been recorded. These may spread from one bather to another, or may be caused by contaminated water and unsanitary bathing appurtenances such as walkways, towels and suits.

Medical literature on other diseases incurred in swimming pools is scarce. Surveys from time to time by public health organizations, both national and state, have yielded little concrete information and few authenticated case histories. State health officials, polled in 1955 by the American Public Health Association, reported no authenticated cases of disease of swimming pool origin in 45 of the 48 states and one territory.

Occasional cases of skin eruptions on the bodies of bathers have been observed. In Wisconsin and Michigan studies have been made of the incidence of "swimmer's itch," or schistosome dermatitis, which has affected bathers in waters infested with Schistosoma haematobium, the invertebrate host being small snails of several genera.

From reports in the recent literature it appears that abrasive accidents in swimming pools have given rise to a new clinical entity known as swimming pool granuloma. Such accidents may occur in diving, in underwater swimming, or in getting in and out of the pool. Reports from Europe, Canada and various parts of the United States indicate widespread geographic distribution of this entity. The purpose of this paper is to discuss this granulomatous infection and to present four cases, believed to be the first reported from Florida or the Southeastern United States.

Review of Literature

In 1951, Hellerström[1] reported six cases he had observed between 1939 and 1949;[2] all were associated with swimming pool injuries, five of the patients having used the same pool, and in all the strikingly uniform clinical features led to a diagnosis of inoculation lupus vulgaris. Some laboratory confirmation of this diagnosis prompted him to call on dermatologists, as well as tuberculosis control officers and sanitary engineers, to give attention to tuberculosis as a water-borne infection.[3] Also in 1951, from Canada, Cleveland[4] reported four cases of possible primary tuberculous infection of the skin complicating abrasions sustained in a public swimming pool in Vancouver. Although laboratory confirmation was lacking, he regarded the clinical and histopathologic appearance of the lesions as highly suggestive of this disease.

Brück,[5,6] in 1951, reported two cases of inoculation vulgaris associated with swimming pool injuries and a third the following year. Nordén and Linell[7,8] made their first report in 1951 and in 1954 published a comprehensive monograph on the subject in which they reviewed the epidemic of 80 cases in one Swedish city, reported the previous year by Herlitz,[9] many of which they had studied. They cited Zettergren,[10] who reported six skin lesions following swimming pool injuries similar clinically to theirs, observed elsewhere in Sweden.

In the United States, Tolmach and Frank,[11] in 1953, described a case observed by them in New York City in which abrasion in a swimming pool accident resulted in an infection diagnosed as granuloma of the skin with tubercle formation. Later that year, Rees and Bennett[12] reported nine cases from San Francisco associated with swimming pool abrasions and diagnosed as tuberculosis verrucosa cutis. They cited Shaw, who in a personal communication reported treating 10 or 12 patients with granulomatous lesions, all of whom sustained abrasions in a swimming pool in Seattle. They estimated 26 to 28 cases had been recognized on the North American continent by 1953. From Texas in 1954, Richardson and Seale[13] reported one case of granuloma following superficial abrasion sustained on the concrete apron of a swimming pool.

*Senior Bacteriologist, United States Public Health Service.
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Etiologic Factors

In one of Hellerström's cases there were acid-fast bacilli in the lesions, and the tubercle bacillus was isolated from a pool used by one of the patients. Although the appearance of the lesions and the histologic findings were consistent with lupus vulgaris, results of guinea pig inoculations and of roentgen examinations of the chest were negative. In four of the cases, however, there was slight enlargement of the regional lymph nodes, and in all six of the patients the tuberculin reaction was positive although in five it had been negative before the injury. Hellerström[1-3] was convinced that the lesions in his cases were manifestations of primary tuberculosis of the skin because of the absence of tuberculous foci elsewhere and the conversion of the tuberculin reactions. In Cleveland's cases, no acid-fast bacilli were demonstrated in the lesions, but clinical and histologic evidence suggested tuberculosis of the skin. Cleveland[4] suggested the possibility of contamination of the swimming pools by persons with incipient or minimal tuberculosis of the lungs or genitourinary tract. In commenting on the reports of these authors in 1952, Sulzberger and Baer[14] questioned the etiology and commented that the smegma bacillus, which may be present in pools in large numbers, produces a "tuberculoid type of response."

In their monograph, Linell and Nordén[8] in 1954 summarized previous studies and concluded that Koch's bacillus was not the etiologic agent. They isolated a previously unrecognized Mycobacterium similar to Koch's bacillus, which they named balnei. They were able to isolate this gram-positive, strongly acid-fast organism from both the water and the walls of the swimming pool associated with the epidemic and to prove conclusively by laboratory studies on animals and on human volunteers that Mycobacterium balnei was the causative organism. By rubbing cement from the pool wall into the skin of a rabbit, they were able to produce tissue changes similar to those in the human lesions. They observed in some preparations a tuberculoid structure, sometimes with central fibrinoid necrosis. Although staining revealed no organisms, in cultures acid-fast organisms were indistinguishable from tubercle bacilli. The organisms were only slightly pathogenic for guinea pigs, but in mice intraperitoneal injections produced ulcerations along the tail and swellings and ulcerations of the feet that were filled with acid-fast bacilli. Histologically, most of the lesions were granulomatous, acid-fast bacilli being demonstrated in but one case.

In the cases of Zettergren,[8,10] attempts at demonstrating tubercle bacilli were unsuccessful. This author regarded Mycobacterium marinum, or a closely related species, as the probable etiologic agent, but Linell and Nordén[8] isolated Myco. balnei from lesions and from the pool. Mycobacteriosis balnearea was the name given to the syndrome.

In the case reported by Brück[6] in 1952, the clinical diagnosis of lupus vulgaris was confirmed when tubercle bacilli were cultured from the lesions and subsequently produced tuberculosis in guinea pigs. These findings led Brück to conclude that mycobacteriosis balnearea, or "so-called swimmer's lupus," could be caused by Mycobacterium tuberculosis and by Myco. balnei. He regarded the former as the responsible organism in his 1952 case and the cases of Hellerström,[1-3] and the latter in the cases of Linell and Nordén[8] and Zettergren.[8,10] After reviewing all available reports of cases, Hellerström[3] concurred in Brück's conclusion regarding similar clinical manifestations caused by different etiologic agents, but he rejected Myco. balnei as the responsible agent. He thought that the organism isolated by Linell and Nordén[8] might be a mutant of Myco. tuberculosis, or that it might be Myco. marinum, as Zettergren[8,10] suggested.

Tolmach and Frank[11] described the lesions in their case as skin granuloma with tubercle formation of unknown etiology. In the cases of Rees and Bennett[12] eight of the nine patients used the same pool, and most of them had a positive tuberculin reaction after the injury. These authors ruled out granuloma due to silicates and deep fungus infections and mentioned the possibility of Myco. tuberculosis or the smegma bacillus as the causative agent, although neither was demonstrated in the lesions or in the pool. In four of the cases guinea pig inoculation yielded no evidence of tuberculous infection. They noted that Shaw[12] did not regard the lesions in his cases as tuberculous because laboratory proof was lacking and because healing took place within a few months. They concluded that these relatively uncommon lesions were benign and somewhat self-limited in nature.

In the case of Richardson and Seale,[13] the histopathologic diagnosis was chronic granuloma, possibly tuberculosis. Regarding the eruption as an infectious process, they suggested that, in

future, cultures be made on several different types of mediums at varying temperatures in an attempt to determine the causative agent.

In their comment on the monograph of Linell and Nordén,[8] Sulzberger and Baer,[15] in 1955, declared it to be a "masterly clarification of a relatively new entity apparently caused by a quite newly discovered acid-fast mycobacterium (M. balnei.)" They considered it virtually certain that this entity was identical with that previously described by Hellerström[1-3] in Sweden, Cleveland[4] in Canada and Rees and Bennett[12] and Tolmach and Frank[11] in the United States. Expressing small wonder that this infection had been diagnosed as a form of tuberculosis in the past, they considered it likely that the uninitiated would continue in this error in dealing with many future cases.

Site and Appearance of the Lesion

Since abrasions incurred in swimming pools usually involve the bridge of the nose, the elbows or the knees, these were the common sites of the lesions in the cases reported in the literature. In Hellerström's cases the patients were males ranging in age from 14 to 29 years, and the eruption developed on the bridge of the nose three to six weeks following the abrasion, which in most cases had healed. The lesions were soft, reddish brown papules similar in diascopic appearance to lupus vulgaris; some were crusted and coalesced. They varied in size "from a pinhead to a spilt pea."

Linell and Nordén[8] described the lesion in their cases as of spongy consistency and located on the outside of the elbow in almost every case. It was slow-growing, became ulcerated, eventually crusted with thick secretion developing under the crusts, and healed spontaneously in six to 24 months. When purulent infection was a complication, the healing process was especially prolonged. A bluish red, soft scar remained, and there was scaling of the surrounding skin.

In the case reported by Tolmach and Frank[11] the site of the abrasion was the bridge of the nose, and the lesion, approximately 2.5 cm. in diameter, in general resembled those described by Hellerström.[3] It was a dull reddish brown granulomatous eruption with distinct margins, and diascopic examination revealed yellowish brown nodules. The entire lesion had a definite firmness and was studded with papulopustules. The patients of Rees and Bennett[12] ranged in age from eight to 20 years, and all but one used the same pool. The site of the lesion was the knee in seven, the right index finger in one, and the front of the ankle in one; in Shaw's patients it was the knee, elbow and nose. These authors pointed out that the lesions in their patients differed sharply in one respect from those of Hellerström,[1-3] Cleveland,[4] Brück[5,6] and others in that they were typical clinically of tuberculosis verrucosa cutis while those previously reported were of the lupus vulgaris type. They stated that lupus vulgaris is a rarity in California. Hellerström, they noted, stressed the fact that his cases were not those of ordinary lupus vulgaris but seemed to result from a superficial inoculation of the skin with the tubercle bacillus, bypassing a violent primary tuberculous complex of the skin. In addition, they thought it should be emphasized that the lesions reported by Hellerström[3] occurred on the nose and were clinically those of lupus vulgaris while those reported by Nordén and Linell[7] and Zettergren[10] were on the elbow and were ulcerated. The lesion was on the elbow in the case of Richardson and Seale.[13]

Treatment

Irradiation with ultraviolet light, Finsen lamp exposures, oral calciferol, and in one case p-aminosalicylic acid were the therapeutic measures employed by Hellerström.[1-3] The lesions healed in three to nine months. In Cleveland's cases the lesions responded to roentgen therapy within three to five months, although destruction with solid carbon dioxide was necessary for residual lesions in one case.

Rees and Bennett[12] used calciferol by mouth, conservative roentgen therapy and, in two resistant cases, isoniazid, which promptly cleared the lesions; healing took place within three to six months. After there was no improvement on treatment with calciferol for five weeks in their case, Tolmach and Frank[11] changed to Promin (di-sodium p, p-sulfonyldianiline-N, N′- diglucoside disulfonate) jelly applied locally for five weeks with good response and then to lotio alba with healing complete five months later. In the case of Richardson and Seale,[13] destruction of the lesion by curettage and electrodesiccation resulted in healing with the formation of a soft pliable scar.

Report of Cases

Case 1. — A 12 year old boy was first seen on Oct. 5, 1956, complaining of a "sore" on his left knee present "all summer" since scraping his knee getting out of a

swimming pool in June. The lesion appeared to heal spontaneously, to be followed about three weeks later by the appearance of several "red bumps," each about the size of a pea. These had grown and coalesced to form a nodule 2 cm. by 1 cm. (fig. 1). The summit of each nodule was ulcerated to varying depths and the tops were crusted. A punch biopsy was obtained and sent directly to the bacteriology laboratory for direct smear and culture. No pathogenic fungi or acid-fast organisms were found on October 5 or November 11. A tuberculin test on this date was negative. From a third culture on December 10, however, an acid-fast rod similar to the tubercle bacillus was isolated. It did not grow at 37 C. on Löwenstein-Jensen medium, and guinea pig inoculation was negative. This organism was identified by the bacteriologist as M. balnei. A repeat tuberculin test on the patient was now positive.

Topical treatment consisted of application of wet compresses of Burow's solution 1:30 for thirty minutes twice daily and the application of an antibiotic ointment dressing at bedtime. The patient was given Dihydrostreptomycin one-half Gram intramuscularly daily for five days and during the succeeding three weeks two other courses of this drug were given for five days without effecting any change in the lesion. A punch biopsy was obtained from the lesion on October 19, and the microscopic picture described by one of us (N.A.M.) was as follows:

"Specimen consists of a disc-shaped, grayish white, solid piece of tissue 4 mm. in diameter and 3 mm. thick. Sections show a covering of mature squamous epithelium. In some areas the epithelium is thickened. An occasional epithelial downgrowth is present, but these areas are fully mature. Just beneath the epidermis is a circumscribed mass of neutrophiles. Additional scattered neutrophiles are seen in other areas. A few epithelioid cells are seen at the edge of the neutrophilic mass, but no definite tubercles are seen and no foreign body type giant cells can be demonstrated. Examination of the tissue under oil immersion fails to show any parasites. The histologic picture and location are suggestive of the so-called "swimming pool" granuloma. There is no evidence of malignancy.

"Diagnosis: Knee: Inflammatory Granulomatous Reaction, benign (compatible with swimming pool granuloma.)"

On October 30, a smear and culture taken directly from the raw surface of a papule resulted in the isolation of several organisms of the Aerogenes group, a hemolytic staphylococcus aureus and an additional strikingly acid-fast organism finally identified as Mycobacterium balnei. Thus, the diagnosis was established of our first case of swimming pool granuloma. On November 19, isoniazid was started orally in a total daily dose of 8 mg. per kilogram of body weight subdivided in three doses in milk after meals. This therapy was continued until November 30, after which time the patient was lost from our observation. During a conversation with the boy's father it was learned that without further treatment the lesion was still present and approximately one-fourth its maximum size by March 1, 1960, nearly four years after it was incurred.

Case 2.—A 15 year old white girl was first seen on March 19, 1958, because of a granulating lesion of seven months' duration situated over the extensor surface of the left knee. The lesion was approximately the size and shape of a kidney bean, spongy in consistency and covered with a purulent discharge. This nodule covered the site of a superficial abrasion sustained on her left knee as she climbed out of a swimming pool "late in August" 1957. Punch biopsy was described by the pathologist as follows:

"Specimen consists of a single, disc-shaped, grayish brown, solid piece of tissue 4 x 4 x 1 mm. Sections show the covering epithelium to be thickened and cornified. There is some hypertrophy of the epithelial pegs. The basement membrane is poorly demarcated in some areas. The deeper portions contain numerous leukocytes. A few

Fig. 1.—Swimming pool granuloma of approximately four years' duration on the knee of a 12 year old boy.

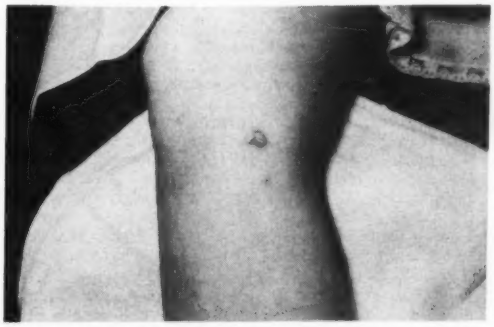

Fig. 2.—Swimming pool granuloma of 7 months' duration on the knee of a seven year old girl.

circumscribed masses of epithelioid cells are seen. There is no evidence of malignancy.

"Diagnosis: Right Knee: Inflammatory Granulomatous Reaction, benign (compatible with swimming pool granuloma.) Tuberculin test was negative."

The patient returned to the office for treatment at irregular intervals and received 250 mg. of streptomycin intramuscularly on April 15, April 29, and May 2. On May 16, 250 mg. of Chloromycetin was given intramuscularly. No other treatment either local or systemic was given. On June 10, the child's mother telephoned that the lesion was completely healed with a barely perceptible scar.

Case 3.—An 18 year old college student was seen on Dec. 23, 1958, during his Christmas vacation because of a crusted plaque on the flexure surface of the left forearm.

Fig. 3.—Microscopic picture of swimming pool granuloma. Note hyperkeratosis, parakeratosis and infiltrates in the corium composed of "epithelioid" cells intermixed with round cells and tendency to tubercle formation.

He stated that in July 1958 he had sustained a "scratch" or a "rope burn" while playing water polo in a pool. The lesion was crusted for several months and would not heal. When first seen on December 23 the boy showed a crusted plaque the size of a five cent piece. There was a central depression or crater 4 mm. in diameter containing a small quantity of cloudy fluid. The borders were raised, translucent, and of soft spongy consistency. Under local infiltration anesthesia the entire lesion was easily removed by curettage and fixed in formalin for laboratory examination. The microscopic findings were typical of swimming pool granuloma. Healing with a smooth noncontracted scar was complete in four weeks.

Case 4.—A child was seen for the first time on Jan. 16, 1960, at the age of six and one-half years. She had been swimming in a public pool only one time in her life on March 31, 1957, but habitually swam in a makeshift swimming area in the St. Johns River near her home in an area protected by the arrangement of large stones and pieces of old cement blocks.

For approximately seven months prior to the first examination of the child her mother had noted "two small bumps" on the extensor surface of her right knee, first seen as two small papules each a little larger than the head of a pin. On Jan. 16, 1960, each of these papules was approximately the size of a pea. The surrounding integument was scaling. The papules were translucent and crusted; beneath the crust was a crater 3 mm. in diameter containing a fairly thick clear fluid apparently of high viscosity as it did not flow over the edge of the crater. Swimming pool granuloma was suspected. The child returned on February 6, by which time the two papules had coalesced (fig. 2). The patient often picked off the crust, which promptly reformed. At this stage a punch biopsy was obtained on February 6, and was described as follows:

"Specimen consists of a single, solid, grayish brown piece of tissue 4 x 3 x 1 mm. Sections show the covering epithelium to be cornified. There is some downgrowth of the epithelial pegs. Some follicular plugging is noticed. An occasional mass of epithelioid cells forming tubercle-like structures is seen. Rather numerous leukocytes of all types are present beneath the surface. An occasional multi-nucleated giant cell is seen. There is no evidence of caseation. Acid-fast infection is to be considered, but I would favor the diagnosis listed below. There is no evidence of malignancy.

"Diagnosis: Right leg: Swimming Pool Granuloma, benign. Direct smear was negative for acid-fast organisms; culture is in progress."

Comment

Two decades ago, the late Dr. H. Marshall Taylor,[16] in reporting his extensive research here in Florida on the health hazards of swimming, wisely observed: "Man is a terrestrial animal and contracts infections in water. . .that run the gamut of pathologic conditions from the innocent circumscribed furuncle to a disease condition of the mastoid." He referred to the field of the otorhinolaryngologist, but his statement has wider application. Mycobacteriosis balnearea appears to be a new hazard which should evoke year-round interest in Florida.

MICROSCOPIC PICTURE.—The cutaneous lesions are located mainly in the corium. The changes seen in the epidermis are probably secondary. Some lesions show moderate hyperplasia of the epidermis with acanthosis. The surface is frequently covered with parakeratotic scales. Sometimes the epithelium has been replaced by fibrin crusts containing polymorphonuclear leukocytes. The epithelial changes are not sharply demarcated but gradually merge with the surrounding normal epidermis (fig. 3).

The corium is usually the site of widespread inflammatory changes which are located in the region just beneath the epidermis and seldom extend below the level of the sweat glands. In a few cases, however, the lesions have broken deeper into the corium.

The lesions themselves are characterized by varying sized infiltrates, dominated by fairly large cells with vesicular nuclei containing nucleoles. These cells have abundant, pale-staining, foamy cytoplasm and vary widely in shape; polygonal, stellate and spindle-shape cells are seen. The large histiocytes or "epithelioid" cells are often intermixed with round cells, mainly lymphocytes. Polymorphonuclear leukocytes and fully differentiated plasma cells are rare. Eosinophil leukocytes are extremely rare. Staining with methylene blue will bring out some scattered mast cells with metachromatically staining violet granules. On the other hand, a fairly large number of giant cells may be

seen. Many of them are of the Langhans type, but smaller giant cells with only a few nuclei, usually located near the margin, are also often seen. The cytoplasm of the giant cells is foamy and pale-staining. Of all the cytological elements, the histiocytes and round cells are by far the most common (fig. 4). They appear in varying proportions, but the histiocytes show a marked tendency to form accumulations which give the inflammatory process a granulomatous character. The epithelioid cells sometimes appear in rounded, discrete, tubercle-like foci (fig. 5). In addition to these inflammatory changes, more or less characteristic of what is generally known as granulomatous inflammation, are infiltrates of histologically nonspecific character. For instance, cellular infiltration around the superficial vessels in the corium or around sweat glands is common. The endothelial cells of the vessels in the center of these infiltrates are swollen.

MACROSCOPIC MORPHOLOGY OF MYCOBACTERIUM BALNEI.—The morphology of the colonies has been very regular on Löwenstein-Jensen medium with incubation at 31 C. Typical colonies appear after nine to 12 days. They are whitish yellow, moist and glistening, rather flat with a central elevation. The edge is slightly irregular. Sometimes radiation folds are noted and sometimes the flattening out occurs in the shape of low terraces. Both "rough" and "smooth" colonies have been found.

MICROSCOPIC MORPHOLOGY.—This is a strikingly acid-fast organism. It is a slender rod, closely resembling the tubercle bacillus, measuring 1.5 to 2.5 microns in length and 0.2 to 0.4 microns in diameter. The shape may vary, and sometimes small, almost coccoid forms have been observed. Occasionally the rod is slightly bent.

PROPHYLAXIS.—While pool-borne diseases are encountered with relative infrequence, the growing popularity of public and home swimming pools, reflected by an estimated increase from 10,000 in 1946 to 132,000 today,[17] confronts the physician with new problems in sanitation and health. The rapidly increasing number of residential pools, which are usually made of concrete and which are not under public health control, is potentially not an insignificant factor in raising the country's morbidity statistics. The newly recognized clinical entity of swimming pool granuloma is a case in point. It deserves the considera-

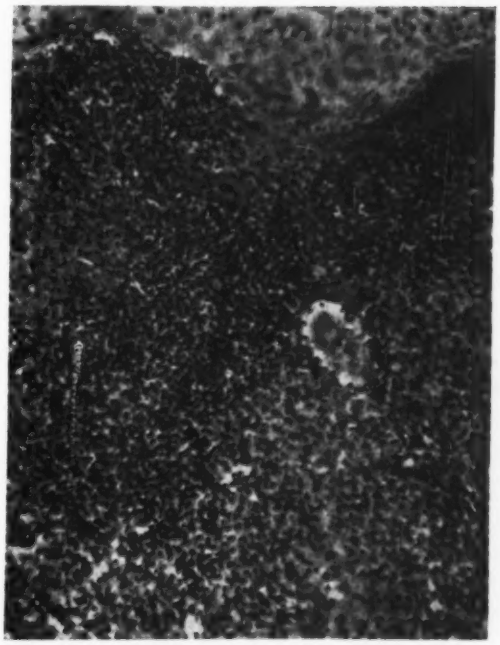

Fig. 4.—Microscopic picture showing typical round cell and epithelioid cell infiltrate in the corium. Note giant cell near center of field.

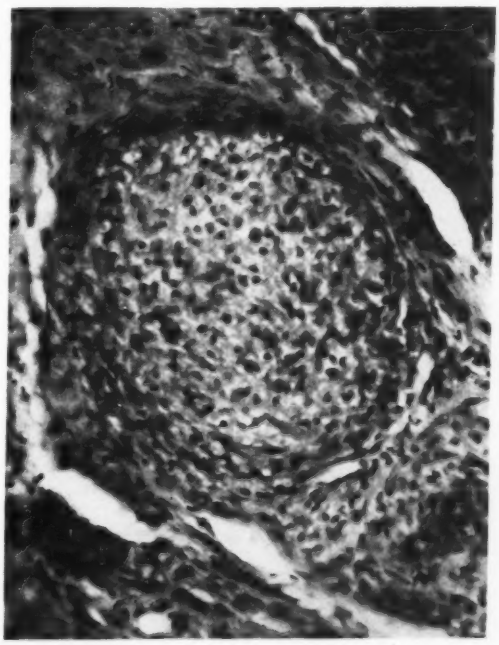

Fig. 5.—Microscopic picture showing tuberculoid structure resembling microscopic lesions of sarcoid. Swelling of endothelial cells of the blood vessels is evident.

tion of general practitioners, dermatologists, otorhinolaryngologists, pathologists and other specialists in the diagnosis of chronic tuberculosis-like or ulcerated lesions of the skin arising at the site of an abrasion sustained in or around a swimming pool.

Greenberg and Kupka,[18] of the California State Department of Public Health, reviewed the limited data available on this disease entity and concluded that the role of swimming pool construction and sanitation may be significant in controlling it. They noted that smooth surface walls and breakfront chlorination had proved effective in curtailing outbreaks. Linell and Norden[8] reported that when the pool associated with the epidemic they studied was rebuilt, the rough concrete walls replaced with smooth tiles, and the chlorination system made more effective, no new cases occurred. In commenting on Hellerström's suggestion that swimming pools be resurfaced with smooth tile, Rees and Bennett[12] remarked appropriately that if this measure does not deal with the causative agent in some specific way, it at least minimizes the possibility of producing abrasions. Other authors[19] also suggested the resurfacing of pools with smooth tile as the best means of dealing with the problem.

Summary

Recent medical literature, principally in the Scandinavian countries and America, directs attention to a new entity connected with abrasive accidents in swimming pools.

Four cases occurring in northern Florida are reported on the assumption that the granulomatous lesions which have been noted following swimming pool injuries in different parts of the world are, despite some variability in clinical characteristics, examples of the same disease.

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1111 Greenleaf Building (Dr. Farrington).
P. O. Box 6296 (Dr. Murray).

Discussion

DR. ALBERT G. LEWIS, Tampa: I certainly have enjoyed Dr. Farrington's excellent paper, "Swimming Pool Granuloma," but since I am neither a dermatologist nor a bacteriologist, my comments, of necessity, will be of a very general nature.

It has been known for many years that mycobacteria include a large number of organisms which are varied in their characteristics and that some are parasites and pathogenic for man or animal, whereas many strains are saprophytes and live in and upon man and animals and in soil and water.

Until recent years the finding of acid-fast bacilli in skin lesions, urine or sputum specimens was considered evidence of infection with human or bovine tubercle bacilli. If, however, these specimens were injected into guinea pigs with negative results, the organisms were considered harmless saprophytes.

We now know that all of the findings shown on the slide, a positive tuberculin skin test, acid-fast bacilli on smear, or even the granuloma with giant cells and caseation necrosis, are not sufficient evidence to prove infection with the human or bovine bacilli. Recently, too, those of us who treat tuberculosis have encountered a number of cases of chronic lung infection due to mycobacteria which mimic tuberculosis in producing a positive skin test and similar histologic and x-ray changes, but which are nonpathogenic for the guinea pig and which, as is the case with chronic skin ulcers due to Mycobacterium balnei, can only be distinguished by appropriate bacteriological methods.

In these cases animal pathogenicity for the guinea pig, or for that matter, any experimental animal, is not necessary to establish pathogenicity to humans.

Several investigators have shown that many different strains of mycobacteria are capable of causing a positive tuberculin reaction, and Palmer, of the United States Public Health Service, several years ago suggested that especially in the Southeastern United States the number of cases of active tuberculosis is too low for the high percentage of tuberculin sensitivity. He postulated at that time that other mycobacteria must be responsible which were as yet unrecognized.

Palmer and his associates also have shown that PPD prepared from 12 different mycobacteria can produce tuberculin sensitivity in the guinea pig, but that when a tuberculin is prepared from each strain of mycobacteria, the reaction to the homologous antigen is generally greater although cross reactions do occur. In instances, therefore, of chronic ulcerative skin disease as presented in this paper, a specific purified protein derivative prepared from the microorganism may be of potential diagnostic value.

Many investigators have noted the nonspecificity of the histology of tuberculosis since various mycobacteria and, in addition, fungi and other agents, may produce an identical pathological picture.

One may summarize by saying that only by detailed bacteriological study can one arrive at a diagnosis, although a history of abrasion and swimming may suggest the possibility of infection with Mycobacterium balnei.

The bacteriological study should include sensitivity studies since most such studies on recently identified mycobacteria reveal resistance to the drugs commonly used in the treatment of tuberculosis, such as isoniazid and PAS, as is the case with Myco. balnei. There, however, is usually a degree of susceptibility to streptomycin and other drugs prepared from the streptomyces suggesting that these drugs are the therapeutic antimicrobials of choice.

In closing, let me emphasize again the nonspecificity of the skin reaction, the histological response to infection, the finding of acid-fast bacilli on smear and the importance of detailed bacteriological studies together with the unimportance of proving pathogenicity for an experimental animal in order to establish pathogenicity in the human.

I am sure that there will be other cases of ulcerative skin lesions due to Myco. balnei recognized in Florida now that their presence is established and if chronic skin ulcers are subjected to bacteriological study routinely.

Again, let me thank Dr. Farrington for allowing me to discuss this very interesting and important paper.

DR. JACK H. BOWEN, Jacksonville: I have enjoyed this very interesting presentation of a rather uncommon, but definitely a clinical entity known as swimming pool granuloma.

I have had one such case which I would like to report and add to the four cases reported by Dr. Farrington. This patient was a 14 year old boy who gave a history of having a superficial abrasion on the right knee received when he fell on the sidewalk. The abrasion healed within four or five days as abrasions usually do, and without difficulty; however, in three or four weeks the patient noticed a nodule had developed at the previously healed site; the nodule persisted and eventually the lesion became ulcerated and drained a serosanguineous fluid when the crust was removed. The lesion had been present for about four or five weeks when I first saw the patient. On biopsy, the specimen was of tuberculoid structure; however, no acid-fast bacilli were demonstrated. A tuberculin skin test gave positive results. The lesion was surgically excised, and the patient was given streptomycin for a period of 10 days. Primary healing of the incision occurred, and there was no further difficulty.

I think there is a very striking similarity in all these cases where a superficial abrasion occurs, rapid healing follows and then a granuloma develops from three to five weeks later. This case differed from the four previously reported cases only in that the abrasion occurred on a public sidewalk instead of a swimming pool. There are several points to reiterate for they are important in pointing out the similarity and differences between swimming pool granuloma and tuberculosis verrucosa cutis of the extremities and lupus vulgaris on the face which it so closely resembles.

First, one sees a positive tuberculin skin test in both diseases for the causative organisms are at least antigenically similar. The skin test may give negative results before the infection and after the infection they may become positive; or, the infection may be superimposed in a person having a positive reaction to a tuberculin skin test.

Secondly, a point of similarity between the two conditions is that few organisms are demonstrated in the tissue and the histologic feature is one of tuberculoid structure.

Difference exists in that swimming pool granuloma is less likely to involve the regional lymph nodes than is tuberculosis verrucosa cutis. Also, culture characteristics of the etiological organisms differ.

Simple eradication of the lesion by excision or desiccation seems to serve well as a form of treatment. Lastly, the swimming pool granuloma has a much better prognosis than the inoculation type of tuberculosis.

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VOLUME XLVII
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Ventricular Septal Defect

An Auscultatory and Phonocardiographic Study of Fifty Cases

LAMAR CREVASSE, M.D.
GAINESVILLE

Accurate diagnosis and clinical evaluation of congenital heart disease are today of great practical importance. The radiographic, electrocardiographic, and catheterization data have been emphasized to the point where diagnosis and absolute values are grossly overstated. The role of clinical diagnosis and evaluation by thoughtful auscultation is neglected. Ventricular septal defect is by far the most common congenital heart lesion and offers no real difficulties in clinical diagnosis. The purpose of this study was to correlate careful auscultation with phonocardiographic, angiocardiographic, electrocardiographic, and catherization data. The total auscultatory spectrum of ventricular septal defect was studied in 50 patients from the first few weeks of life through age 40. Phonocardiograms are shown primarily as graphic illustrations as they do not record sounds not heard by careful use of the stethoscope. Ventricular septal defect can be divided by clinical evaluation into the following groups as determined from these data:

Group I: Ventricular Septal Defect With Small Left to Right Shunt and Normal Pulmonary Artery Pressure

There were 24 patients studied in this group. They were essentially asymptomatic, and the defect was initially detected by the presence of a rasping, grade IV pansystolic murmur, maximal in the third and fourth left intercostal space, which is almost invariably accompanied by a thrill and sparsely transmitted to the apex. Over the pulmonic area is a decidedly separate blowing pulmonary systolic flow murmur (fig. 1a). The pulmonic second sound is normally split and of normal intensity (fig. 1b). The pulmonary artery pressure in this group was usually normal and always under 50 mm. Hg (table 1). The third heart sound at the apex representing rapid left ventricular filling is readily detectable and of moderate magnitude and duration. There was minimal or no cardiac enlargement, and the degree of left to right shunt at the ventricular level was minimal to moderate.

Group II: Ventricular Septal Defect With Large Left to Right Shunt and Moderate Pulmonary Hypertension

With larger defects and increased pulmonary blood flow, pulmonary artery pressure is elevated and closes the pulmonic valve earlier and with more vigor, producing accentuation and narrow splitting of the second heart sound. In this group (table 1) pulmonary artery pressure was elevated and ranged between 25 and 60 mm. Hg with the vast majority in the range of 40 to 60 mm. Hg. The third sound is prominent, and its duration and magnitude roughly parallel the degree of shunt. The systolic murmur remains essentially unchanged. There is usually combined ventricular hypertrophy by physical examination and the electrocardiogram. There is an appreciable left to right shunt.

Group III: Ventricular Septal Defect With Moderate to Large Left to Right Shunt and Severe Pulmonary Hypertension

There were 11 patients in this group. When pulmonary artery pressure exceeded 60 mm. Hg, the pulmonic second sound was markedly accentuated and single (table 1). The systolic murmur varied in intensity with the magnitude of the systolic pressure gradient between the left and right ventricle. As right ventricular pressure rises in response to pulmonary hypertension, the systolic murmur becomes of less intensity. The third heart sound again parallels the degree of shunt (fig. 2b). There may be solely right, left, or combined ventricular hypertrophy, depending on the degree of shunt and pulmonary hypertension.

Group IV: Ventricular Septal Defect With Balanced Shunt and Severe Pulmonary Hypertension (Eisenmenger's Physiology)

With very severe pulmonary hypertension, right ventricular systolic pressure becomes equal to or exceeds left ventricular and the shunt be-

Presently Florida Heart Research Investigator and Assistant Professor of Medicine, College of Medicine, University of Florida, Gainesville, Fla.

comes balanced, bidirectional or reversed. When right ventricular pressure equals or exceeds left, this obliterates the interventricular systolic pressure gradient and the systolic murmur may disappear, producing a "silent ventricular septal defect." The pulmonic second sound is now strikingly accentuated and single, and the murmur of pulmonic insufficiency may be present (fig. 2c). There is nearly always severe right ventricular hypertrophy. Atrial septal defect, "reversed" patent ductus, and primary pulmonary hypertension may, however, produce the same clinical picture in advanced stages.

Atrioventricularis Communis

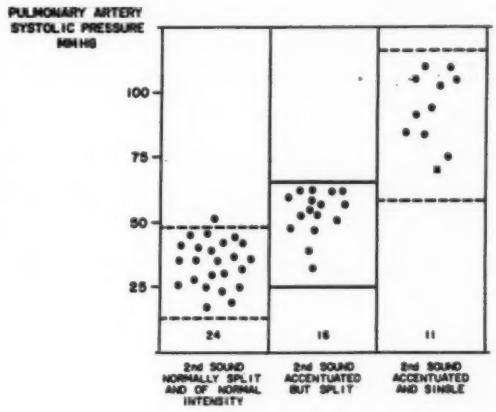
Three patients in this study had a combined atrioventricular communication. This differs in many respects from isolated ventricular septal defect. The murmur, unlike ventricular septal defect, is usually widely transmitted to the apex and may be maximal in this area (fig. 3). A congenital cleft deformity of the mitral valve is commonly associated with this defect and may produce the typical murmur of mitral regurgitation and/or stenosis (fig. 4). Pulmonary hypertension is commonly present, producing accentuation of the pulmonic second sound in relation to the severity of the pulmonary hypertension. Left axis deviation and first degree atrioventricular block (fig. 5) are prominent electrocardiographic features.

Ventricular Septal Defect Simulating Pulmonic Stenosis and Patent Ductus Arteriosus

Occasionally in children, the murmur of ventricular septal defect may be diamond-shaped and maximal in the second left intercostal space, simulating pulmonic stenosis (fig. 5). The pulmonic second sound, however, is usually pathologically split in pulmonic stenosis. A pulmonary artery click and the late systolic murmur overriding the aortic component of second sound are characteristic. Diminution of pulmonary blood flow in contrast to increased pulmonary flow with ventricular septal defect is present.

Ventricular septal defect may mimic patent ductus arteriosus when loss of support of the aortic cusp by a high ventricular defect produces an associated aortic regurgitation. This creates a to and fro murmur closely simulating patent ductus, but can be differentiated phonocardiographically by the graphic relationships in that the bulk of the systolic murmur of patent ductus is

Table 1.—The distribution of relationships of closure of the aortic and pulmonic components of the second heart sound to pulmonary artery systolic pressure (see text).



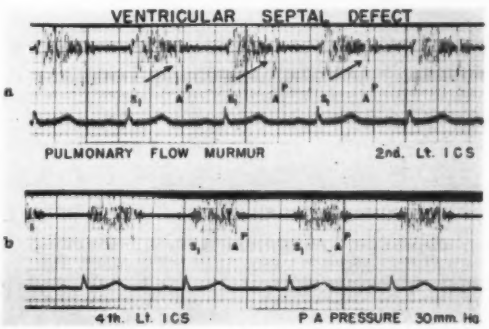


Fig. 1.—S_1=first sound. A, P = aortic and pulmonic components of second sound.

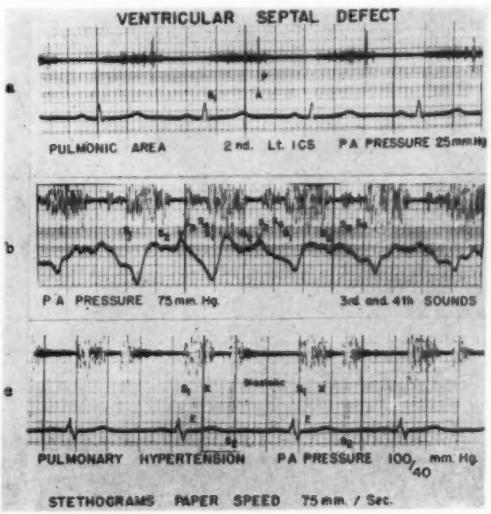


Fig. 2.—S_3, S_4= third and fourth heart sounds. X = contact sound. E = pulmonary artery ejection sound. S_2 = single second sound.

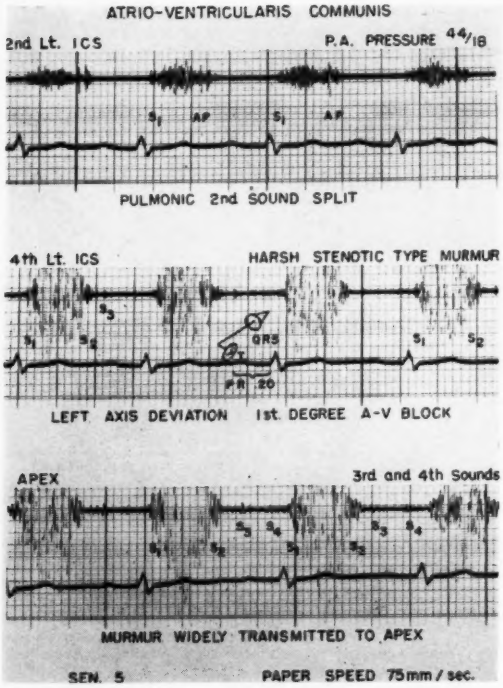


Fig. 3.—S_1=first sound. S_2=second sound. A, P= aortic and pulmonic components of second sound. S_3, S_4 = third and fourth heart sounds.

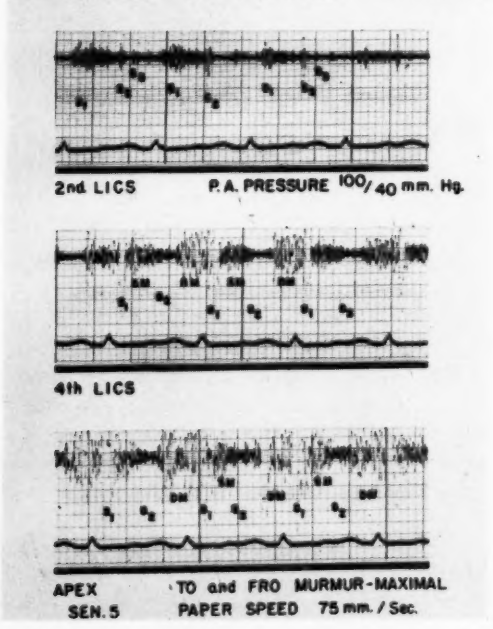


Fig. 4.—S_1, S_2=first and second sounds. S_3=third sound. SM = systolic murmur. DM = diastolic murmur.

delayed and will override the second sound completely. Likewise, when patent ductus is atypical and presents with a systolic murmur alone, it may be of considerable intensity and simulate ventricular septal defect. But again, it is late and grossly overrides the second sound.

Ventricular Septal Defect with Pulmonic Stenosis or "Acyanotic Tetralogy of Fallot"

Five patients, or 10 per cent of the entire group studied, had an associated infundibular and/or valvular pulmonic stenosis without cyanosis, a condition confused with isolated ventricular septal defect. Because of the obstruction to right ventricular outflow, however, the pulmonary artery pressure is low, and pulmonic valve closure is considerably delayed beyond 0.06 second, or may not be detected. Furthermore, the intensity

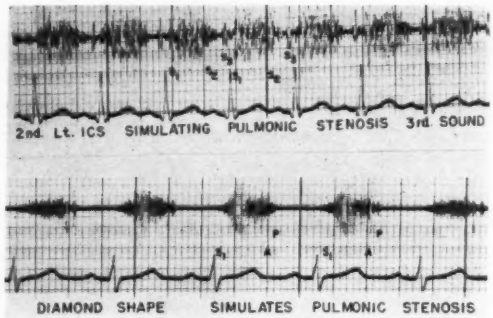


Fig. 5.—S_1, S_2 = first and second sounds. S_3 = third sound. A, P = aortic and pulmonary components of second sound.

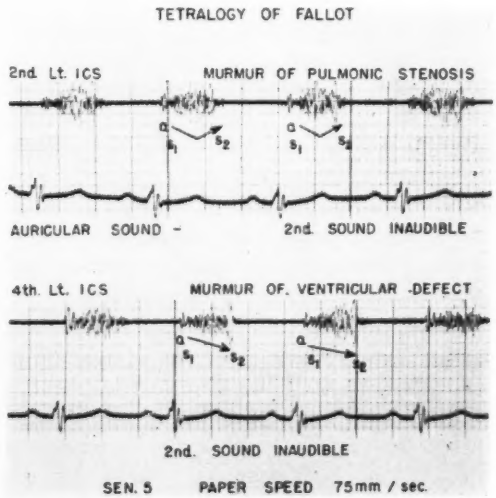


Fig. 6.—A = auricular sound or S_4. S_1, S_2 = first and second sounds.

of the systolic murmur is considerably less. By careful auscultation, the stenotic diamond-shaped murmur of associated pulmonic stenosis may occasionally be differentiated from the accompanying murmur of ventricular septal defect (fig. 6).

Summary

There are several points of value:

1. There are two murmurs with isolated ventricular defect. One is related to the defect per se and accounts for the rasping well localized systolic murmur in the third and fourth left intercostal space. The other distinct murmur in the second left intercostal space is related to increased flow across the pulmonic valve.

2. The status of pulmonary hypertension is of extreme clinical and prognostic importance. It can be reasonably assessed by the character of the pulmonic second sound. As long as pulmonary artery pressure is normal, the pulmonic second sound is normally split and of normal intensity. When pulmonary artery pressure is in the range of 40 to 60 mm. Hg, the pulmonic second sound is narrowly split and accentuated. When pulmonary artery pressure exceeds 60 mm. Hg, the pulmonic second sound is markedly accentuated and single. With severe degrees of pulmonary hypertension interventricular pressures may become equilibrated and the murmur of ventricular septal defect may disappear, producing Eisenmenger's physiology.

3. A third heart sound reflecting rapid ventricular filling is commonly present at the apex. Its magnitude and duration parallel the degree of the shunt.

4. Atrioventricularis communis can be suspected when the murmur of ventricular septal defect is maximal at the apex or the auscultatory findings of mitral regurgitation and/or stenosis are associated with it as a congenital cleft deformity of the mitral valve is usually present.

5. Ten per cent of patients suspected of having isolated ventricular septal defect will have an associated valvular and/or infundibular pulmonic stenosis without cyanosis. Careful auscultation will reveal the stenotic diamond-shaped murmur of associated pulmonic stenosis, and the pulmonic second sound is usually not detectable or considerably delayed.

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College of Medicine University of Florida.

The Last Word . . .

(Continued from page 379)

submitted such a plan for approval of the Federal Department of Health, Education and Welfare.

This would require no additional state appropriation by the state and provide 75 per cent relief to the counties in medical welfare costs to those 65 years of age and over, which are estimated to comprise over one-half of the needy sick of all ages now receiving medical care from the county and state. (One hundred per cent counties participation required to obtain federal matching funds.)

The issue of using federal matching funds in state programs for the needy sick was decided in 1935 with establishment of the Social Security law. The challenge now is to provide good comprehensive medical care for the needy sick under existing law and institutions motivated by Christian principles and for the preservation of human resources which will render this emotionally appealing issue useless to those who would use it as a vehicle to carry us on the socialist road to destruction.

A Preliminary Report of Cardiovascular Surgery Under the Florida Crippled Children's Program

WILLIAM R. STINGER, M.D.
TALLAHASSEE

In 1956, the Florida Crippled Children's Commission began to provide diagnostic services and treatment for children with operable heart conditions whose parents could not afford private care. Over 500 children with signs and/or symptoms of possible heart disease have been referred to Commission clinics since that time, and this paper describes the program and some of the results obtained.

Prior to 1956, intrathoracic and cardiovascular problems in infants and children were relatively unusual under the Commission program; so the Commission began by appointing a committee to investigate the special needs of these children. The committee, composed of recognized authorities representing appropriate professional disciplines, made recommendations which helped guide the Commission's efforts. These recommendations stressed the team approach to the problem, and it is interesting to note that similar recommendations were independently developed by the Subcommittee on Education and Standards of the Council on Rheumatic Fever and Congenital Heart Disease of the American Heart Association.

Based on incidence and prevalence surveys in Escambia County and Dade County, it was also possible to make a rough projection of the case load. In 1956, it was estimated that there were approximately 6,000 persons in Florida with congenital heart disease, and at least a third of the cases would be operable. As indicated in table 1, this ratio proved fairly accurate. As far as the Commission was concerned, it was further estimated that about 150 cardiac referrals would be served each year, and approximately 50 would be deemed amenable to surgery. Subsequent experience has also confirmed these estimates within reasonable limits. Like most new activities, the Commission's cardiac program served relatively few children in the beginning, but there has been a steady increase in the number of referrals, and the Commission is now serving as many children as its funds permit.

The breakdown of conditions diagnosed and operations performed is shown in table 1. It may seem strange that the Commission observed many more ventricular septal defects than conditions such as patent ductus arteriosus. In considering these statistics, one must remember that this state agency limits its activities to the medically indigent, and diagnosis and repair of a defect

Table 1.—Types of Cardiovascular Conditions Diagnosed and Treated Under Florida Crippled Children's Program, 1956-1959

Cardiovascular Defect	Number of Patients	Number Operated on
Atrial septal defect	47	16
Aortic stenosis	16	4
Coarctation of aorta	15	5
Patent ductus	60	38
Pulmonary stenosis	37	23
Tetralogy of Fallot	62	26
Ventricular septal defect	163	20
Mitral stenosis	1	1
Lesion undetermined	105	0
	506	133

Table 2.—Results of Cardiovascular Surgery Under Florida Crippled Children's Commission Program, 1956-1959

Cardiovascular Defect	Greatly Improved	Improved	Unimproved	Deaths	Totals
Atrial septal defect	9	4	0	3	16
Aortic stenosis	0	3	0	1	4
Pulmonary stenosis	11	12	0	0	23
Patent ductus	23	12	1	2	38
Coarctation of aorta	3	2	0	0	5
Tetralogy of Fallot	8	10	2	6	26
Ventricular septal defect	7	10	0	3	20
Mitral stenosis	1	0	0	0	1
	62	53	3	15	133

such as a patent ductus is usually not as involved or expensive as diagnosis and surgery for conditions such as ventricular septal defects or tetralogies. It is highly probable, therefore, that most of these less complex conditions are examined and treated on a private basis and are not referred to the Commission. This preliminary screening also accounts for the relatively small number of patients with rheumatic heart disease or no heart disease.

As of October 1959, surgery had been performed on 133 of the 401 children with definite diagnoses of congenital heart disease and one patient with rheumatic heart disease. Twenty-six additional patients were scheduled for future surgery. As far as the remaining cases are concerned, most of these were either deemed inoperable by present surgical technics or represented minimal defects where surgery did not seem warranted. The parents of a few patients refused surgery, and several other patients were returned to the care of their private physicians with recommendations for surgery. The Commission has not attempted to follow the latter group.

Table 2 is based primarily on a subjective or symptomatic evaluation of the results of surgery. In some instances, the evaluation is also based on objective hemodynamic evidence including preoperative and postoperative catheterization reports. The Commission plans to obtain objective data on more of these patients over a greater period of time, and these results will be reported at another opportunity. Despite the inherent dangers of conclusions based on subjective information, the Commission is confident that the program is transforming most of these children, previously doomed to a short life of chronic invalidism, into virtually normal useful citizens.

P. O. Box 1508.

The Complete "D. & C."

ALVAN G. FORAKER, M.D.
JACKSONVILLE

Irregular bleeding may come from lesions high in the endocervical canal, inapparent on examination. These may include "in situ" or invasive squamous cell carcinoma. They may be present in patients whose cervices appear normal to inspection. Occasionally fragments of the tumor may be dislodged and included with corporeal endometrium in a standard dilatation and curettage. This happenstance may lead to the diagnosis of cervical carcinoma, even in the absence of a cervical biopsy. This is relying on a weak reed. Many cases of early cervical carcinoma must be missed in this fashion, to the patient's detriment.

When a patient is being subjected to dilatation and curettage of corporeal endometrium, cervical biopsy would involve only a slight increment in anesthesia time, surgical procedure and bleeding. The possible benefits are great, the deterring factors minimal.

From the Pathology Laboratory, Baptist Memorial Hospital, Jacksonville, Fla.

It is urged that cervical tissue be obtained for examination in every instance where corporeal curettage is undertaken for diagnosis or for treatment of irregular bleeding. Possible exceptions might include:

1. Cases with recent negative office biopsy. Office biopsies, however, rarely include endocervical curettage, and may not reveal concealed lesions in the endocervical canal.

2. Cases in which the slight increase in bleeding and in anesthesia time would be of critical importance.

3. Cases with recent negative cytological examination. Cytology is of utmost importance in detection and evaluation of uterine (including cervical) lesions. A positive or suspicious smear can lead to exact diagnosis (by tissue examination) and is of marked value. A negative smear has only cumulative value. It should not prevent further investigations in patients with clinically suspicious signs and symptoms. When smears are properly taken and processed, comparatively few

false negatives will occur in cervical neoplasms. The possibility, however, does exist. If a patient is to have dilatation and curettage of corporeal endometrium, cervical tissue should be examined also.

4. Cases of dysfunctional bleeding, as in infertility studies. One must, however, be sure that endocervical lesions play no part in the patient's disorder. How better can this be accomplished than by biopsy and endocervical curettage? The age group of cervical carcinoma overlaps that of clinically important infertility. Examination of cervical tissue can be of marked importance in these cases.

For optimum diagnostic value, the tissue specimens should be submitted separately as corporeal endometrium, endocervical curettings, and two or four quadrant squamocolumnar junction biopsies.

If examination of cervical tissue is made a part of "the complete D. & C." many patients will benefit.

Summary

As an important measure in the detection of early cervical carcinoma, it is urged that endocervical curettage and squamocolumnar junction biopsy be made a routine portion of dilatation and endometrial curettage, when done for diagnosis or for irregular bleeding.

800 Miami Road.

ABSTRACTS

Treatment of Meprobamate Intoxication with the Use of Methyl Phenidate: CASE REPORT. By David Potyk, M.D. South M. J. 52: 1148 (Sept.) 1959.

The purpose of this paper is to discuss the treatment of cardiovascular depression and collapse, with respiratory depression following an overdose of meprobamate in an elderly woman who attempted suicide. The use of methyl phenidate intravenously resulted in marked improvement with eventual recovery. It appears that this drug may be a useful antidote to meprobamate intoxication and by itself has had few side reactions.

Treatment of Acute Gout With Injection of Colchicine. By Lynn P. Carmichael, M.D. J. A. M. A. 170:1410-1411 (July 18) 1959.

While newer agents are valuable in the treatment of gouty arthritis, colchicine, one of the oldest and most specific pharmaceuticals available, remains the most useful drug in the treatment of this disorder in the opinion of this author. Cochicine injection is now available for intravenous administration. When the drug is given by this method in the indicated dosage, gastrointestinal side effects rarely occur and the response is more rapid than that obtained with the oral method. Four cases are reported which illustrate the diagnostic and therapeutic advantages of intravenously administered colchicine. It is concluded that the intravenous administration of this drug is a rapid, safe, and uncomplicated method of treating acute gouty arthritis. Also, it is of special value as a diagnostic procedure in those cases in which the diagnosis of gout is doubtful, because of its rapid action and the freedom from nausea, vomiting, and diarrhea often associated with oral administration.

A New Method for Identifying Blood of the Portal Circulation. By Winston K. Shorey, M.D., William H. Meyer, M.D., W. A. D. Anderson, M.D., Raymond E. Parks, M.D., and Norman M. Kenyon, M.D., South. M. J. 52:1052-1057 (Sept.) 1959.

This report deals with the methods employed in and the results of experiments pursued in the development of technics for differentiating blood in the portal circulation from blood in the systemic circulation. A concept for detecting the escape of blood from esophageal or gastric varices by introducing an identifiable material into the portal circulation and then finding a significant concentration of it in blood aspirated from the stomach is presented. Identifiable materials were introduced into the portal circulation of dogs by injection into the wall of the lower colon by means

of a modified sigmoidoscope. The injection of fluorescein resulted in portal vein concentrations of dye consistently higher than concentrations in the vena cava. Pathologic study revealed no significant damage to the bowel of dogs in which this technic was used. Radioactive iodine instilled into the lumen of the lower bowel was found to be absorbed just as effectively as that injected into the wall of the bowel. Human experiments demonstrated an increased absorption rate when it was instilled in an alcoholic solution as compared to instillation in physiologic saline. Further investigation is required to determine whether or not these observations can be applied to the practical problem of identifying hemorrhage from a ruptured esophageal or gastric varix. The authors note that the secretion of a high concentration of iodide by the gastric mucosa, as found by other investigators, probably precludes the use of iodine-131 for this purpose.

Correlation of Smoking Habits With Hemolytic Streptococci and Antistreptolysin O Responses. By Milton S. Saslaw, M.D., and Murray M. Streitfeld, Ph.D. Am. J. M. Sc. 237:754-758 (June) 1959.

Epidemiologic studies on β hemolytic streptococci conducted in Miami by the authors gave them an excellent opportunity to investigate any possible relationship of smoking habits of adults to the prevalence of streptococci in the human throat. Accordingly, data on smoking habits were collected at the same time that throats were swabbed for isolation of streptococci and venous blood samples were drawn for streptococcal antibody determinations. Analysis of findings from 1,812 throat swabbings showed that β streptococci were isolated from the throats of present smokers almost twice as often as from nonsmokers or past smokers, a highly significant difference statistically. Group A organisms also were isolated more frequently from smokers' throats, but the difference was not as great. Antistreptolysin O studies (1,611 samples) showed no appreciable differences in titers of serums from smokers when compared with those from nonsmokers. The authors concluded that the higher frequency of streptococcal isolation among smokers than nonsmokers and past smokers may be due to an effect of smoke products on streptococci or on the mucous membrane barrier of the host.

Wegener's Granulomatosis. Case Report: Antemortem Diagnosis and Observations on Effect of Treatment. By Ulfar Jonsson, M.D., and Dewitt C. Daughtry, M.D. Am. Pract. & Digest Treat. 10:47-52 (Jan.) 1959.

Wegener's granulomatosis consists pathologically of a necrotizinz granulomatous process of the respiratory tract, generalized vasculitis and focal glomerulitis. Clinically, it is a fatal disease entity characterized in the early stages by severe rhinitis and paranasal sinusitis often accompanied by pulmonary lesions simulating inflammation or malignancy; the later stages are characterized by a generalized arteritis and renal insufficiency. The diagnosis is seldom made during life. The authors describe in detail an instance of this disease diagnosed nine months antemortem. The presenting symptoms were rhinitis and sinusitis followed by multiple discrete nodules in the lungs. A considerable degree of symptomatic relief of nasal symptoms and clearing of pulmonary lesions followed steroid therapy. Death apparently was due to surgical complications and sepsis following a right lower lobe lobectomy. The literature is briefly reviewed, and the relationship between Wegener's syndrome and other similar clinical conditions is discussed.

Anesthesia for the Aged: A Comparative Evaluation. By Harry E. Danielson, M.D., and J. Gerard Converse, M.D. South M. J. 52:1132-1136 (Sept.) 1959.

A study on the relative safety of spinal versus general anesthesia in the aged patient undergoing an operation on the hip was undertaken with a view to throwing some light on one facet of the total picture of geriatric anesthesia. A group of 90 geriatric patients who had hip surgery was chosen at random for a comparison of operative and postoperative mortality and morbidity. Spinal anesthesia was used in 46 and general anesthesias in 44. The authors suggest that general anesthesia, utilizing the technic of analgesia plus relaxants, offers a more satisfactory method than the use of hypobaric spinal anesthesia.

Members are urged to send reprints of their articles published in out-of-state medical journals to Box 2411, Jacksonville, for abstracting and publication in The Journal. If you have no extra reprints, please lend us your copy of the journal containing the article.

PRESIDENT'S PAGE

The Bright Side

All too often, and particularly when impending legislation points in a direction contrary to their desires, or a candidate for office holds views inimical to the best interests of medical practice, physicians are heard to lament the lack of leadership and political activity on the part of organized medicine as represented in the American Medical Association and the Florida Medical Association.

Legally, the limitations on political activity as outlined in various acts, namely, the Hatch Act, the Corrupt Practices Act, and the New Criminal Code, as relating to medical organizations are these:

1. The American Medical Association cannot legally contribute to or expend funds in support of, or in opposition to, candidates for federal office. (I presume the same law applies to labor unions.)

2. State and county medical societies, whether incorporated or not, are subject to the same limitations.

3. The law prohibits a medical society from: a. Endorsing a candidate, where it involves expenditure of general corporate funds. b. Contributing funds to any candidate for federal office. c. Using medical society letterheads or facilities to advance work in behalf of a candidate. d. Sponsoring any other form of advertising material for a candidate.

4. Individuals forming political committees must not make use of any official position or office which they may hold or occupy in any organization.

The Florida Medical Association and, it is hoped, its component societies lean over backward to stay well within the letter of the aforementioned rulings. On the positive side, medical societies, as organizations, can and should urge their members and others to register and vote.

There is a bright side, however, and it is unfortunate if not astounding that so many of our membership are unaware of an organization formed in 1950, independent of organized medicine, limited in membership to doctors of medicine and their spouses, and openly dedicated to engage in political activity. This group, the Florida Medical Committee for Better Government, arose out of the effective core of physicians and their wives who were instrumental in putting Senator George Smathers in office in 1950, defeating the incumbent, an outspoken advocate of socialized medicine and supporter of the Murray-Wagner-Dingell Bill. This Committee aided, in 1958, the return of Senator Spessard Holland to Washington against the candidacy of the same so-called liberal.

Within the state, even on the local level, this Committee has spoken out and supported candidates for various offices when it was in the best interest of the people to do so.

The objectives of the Florida Medical Committee for Better Government as noted in Article II of its By-Laws are: A. To uphold the tradition of individual liberty and the system of free enterprise. B. To constantly encourage and stimulate physicians toward better and more active citizenship. C. To stimulate and cooperate with similar committees of other groups whose purposes are in harmony with the aims of this body. D. To study the issues of government, records and platforms of candidates aspiring to state and national offices, and to make results of such studies publicly available. E. To cooperate with similar committees in other states.

In an election year, when good men are running for office as well as other men who are obviously opportunists, willing to stop at nothing to obtain votes, it is imperative that physicians, being educated, thoughtful, and far-seeing citizens, take an active part in politics. The Florida Medical Committee for Better Government gives you that opportunity to channel that activity where it can be most effective. Your dues, a pittance, are needed. Your support is even more desirable.

Leo M. Wachtel

THE JOURNAL
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More on the Art of Medicine

I do not love thee, Doctor Fell.
The reason why I cannot tell;
But this alone I know full well,
I do not love thee, Doctor Fell.

These words are thought-provoking today just as they were when written by Tom Brown, student at Christ Church, Oxford, nearly 300 years ago. Ofttimes one cannot express articulately the reason for his dislike or dislove of a person—a physician—but in the last analysis it frequently is the attitude of the disliked.

If a physician saves a life but does it in an impersonal, cold, ungracious manner, he is not apt to get much thanks. On the other hand, if a physician loses a patient by death, and his attitude has been one of solicitude, kindness and graciousness, members of the family are more apt than not to defend him and be grateful to him.

Two cases in point are that of an elderly, diabetic woman who lived through peptic ulcer perforation with peritonitis for 18 hours before she was seen professionally, and that of a middle-aged man who died unexpectedly while receiving antibiotics and general supportive care for lobar pneumonia. A review of the two cases gave rather convincing proof that the life of the first patient was saved by the professional skill of the doctor. Yet, the patient and the members of her family were not only unimpressed, but were even a little unhappy, for the doctor in charge had been gruff, had not taken time to explain the details of the case and had not made them understand what he was trying to accomplish. In the second case, however, the surviving members of the family could not seem to say enough by way of praise of the physician. He had been thoughtful and kind; he had explained the illness to those concerned and had given unsparingly of himself.

It might be remembered that most physicians in the horse and buggy era held the love, affection and deep respect of their patients even though they knew much less scientific medicine than we know today. They had much less they could say, but they knew when to talk and how to say it.

The medical profession has something to talk about, something to give and something to sell which is priceless, but frequently a problem arises —how much to charge?

Recently, in a talk before the Florida Society of Internal Medicine at its annual meeting in Jacksonville, a distinguished lay speaker com-

plained that he could not understand why he could not get a firm estimate of charges for professional services on health insurance policies prior to rendering of the services. It should hardly be necessary to remind our lay friend that services depend upon many intangibles that frequently cannot be foreseen. Not until a good history and thorough physical examination have been obtained can the physician predict how many laboratory, x-ray and other clinical studies will be needed and sometimes not then. Consequently, he frequently cannot foretell how much professional service will be appropriate to the individual case. In the interest of good public relations, perhaps members of the medical profession will want to agree upon a firm estimate for group health insurance policies with the hope that the total will average up satisfactorily.

A wise professor once advised one of the Editors that as much as possible he should deal with those people with whom he could establish rapport, and that he should make every effort to fill his practice with people whom he liked and who liked and appreciated him. He pointed out that it does not matter whether such are rich or poor, well-to-do or ordinary; funds will be forthcoming and fees will almost take care of themselves. On the other hand, there are people in all walks of life who cannot be pleased, who seem to look for faults and for someone to blame for their unhappy plight. An older lay friend, rich in knowledge of worldly affairs, put his advice more succinctly—if negatively: "You can't get anywhere dealing with sorry people."

If any phyiscian should doubt the importance of attitude in the office and at the bedside, let him try to picture a patient in real need of medical aid and advice, and then visualize what a difference may be made by the appearance of a physician with confidence and keen interest in the over-all medical problem at hand. Such a physician will want to charge for his services appropriate to the patient's station in life and his ability to pay. There will be no overcharge, for such services are quite priceless.

The Decline of American Medicine

The disintegration of American Medicine as it is now practiced may be expected to occur at an accelerated rate. Proof is everywhere, but no better evidenced than in the poor attendance and lack of accomplishment at most County Medical Society Meetings. Reasons for this disinterest are many, but cardinal among them are apathy, the division of physicians amongst themselves, implicit faith in the American Medical Association and its officers, and a general intransigence of thought on all paramedical problems.

Lucrative practices and the social prestige of the medical profession have removed the stimulus for change so necessary in an evolving social cosmos. The benign contentment of past accomplishment is merging into a paralyzing apathy toward any but professional problems. As medicine has become more complex and has borrowed increasingly from other scientific disciplines, divisions have occurred which are now pitting physician against physician whether it be the solo practitioner against a group, academists against practitioners, teaching institutions against organized medicine locally, and above all, supervisory councils, which physicians have developed, against physicians themselves. The various specialties whose only reason for existence is the attainment and application of wider knowledge are continually sniping at each other. Each specialty is more interested in protecting its own vested interest than in the preservation of medicine as a whole. Indeed, medicine is now a house divided.

Once the average American physician has become a Member of the American Medical Association, he believes that each and every one of his nonprofessional problems will be magically solved. One need only review the events of recent years to see how false this belief is and how insecure the position of the American physician has now become. A few years ago, Medicare appeared on the horizon. That it exists in its present state is no kudos for government or American Medicine. Recently, the Federal Employees Insurance Program has developed. The average

physician has no knowledge as to the functioning of this far-reaching legislation and how it will affect him. More recently, health insurance for the aged has been in the forefront. In each of these instances, the American Medical Association has run a scared race. In each, we have come off a second best. The reasons are simple. Every problem is given the same trite treatment and answer, namely: (1) Let's keep our own house clean, that is, admit to the public we are probably wrong, but we are going to improve. (2) Let the existing mechanisms take care of the problem, or why develop an "H" bomb when we have cannon balls. (3) Let us educate the public, or tell the people they really do not want social security and other benefits. (4) Have a five or eight point program of generalities which do not solve the problems—after all we are all against sin and that is enough.

It has been unfortunate that in recent years some of the leaders of American Medicine have been more concerned with their own aggrandizement than with the solution of the problems of organized medicine. The oratory of a recent American Medical Association president could not be surpassed, but it was ineffective. The fact that he challenged his adversaries to debates which never occurred did little to help our lot. At this time, we need no silver-tongued high school debaters, but thinkers! It is a sad commentary that medical politics is usually run by default. Practically anyone who is willing to attend meetings, support the status quo and never take a stand which will make members commit themselves on any issue is certain to get to the top. The American Medical Association on paper is a very democratic organization. In practice, however, the state delegates rarely change and rarely do they oppose or propose anything that has not been devised in the Chicago headquarters.

We can no longer afford officers to represent us in our own county societies or in Chicago who are apathetic or ineffectual. We need leaders who are willing to take a stand—a point of departure —whether they make enemies or not. It is time to stand and be counted.

Unless we as physicians begin to take an active part in our local organizations and elect delegates to do our bidding, we deserve the fate of our colleagues in other parts of the world. The problems which face American Medicine today are numerous and have been engendered by dynamic socioeconomic changes with which the physician alone cannot cope. First, we must face them. Then we must delineate and define them before we can solve them. Adequate care for the aged, indigent care, how best can the physician deal with a third party in medicine whether it be labor, industry, insurance carrier or government —these are merely facets of the multiple and as yet really unknown and undefined problems which beset us.

Thus it becomes the urgent duty of the American Medical Association in conjunction with its component societies to organize and conduct a study by a commission composed of the finest sociologists, economists, and political scientists that money and a virgin field for inquiry can entice. These select elite must meet with the heads of labor, industry, government and medicine in an attempt to find what the actual medical needs of the American public are and how best to meet them. We cannot enter into this study with a preconceived idea as to what our conclusion must be any more than an honest research worker begins a study attempting to prove a point. We must also discard the juvenile thought that American Medicine must exist in its present form such as it has for the past century or more and expect everything else in this land to conform to it.

Naturally, it will be prudent and atraumatic to solve our problems, if possible, within our present framework of medical practice. There is nothing radical nor socialistic with the foregoing realistic approach to the solution of our organizational and socioeconomic problems. The methods suggested are those we use daily in our every research center. Unless the American physician is willing to contribute a small amount of time, effort and money, the decline of American Medicine will rapidly enter the abyss of lay-controlled medicine. The best solution of our paramedical problems lies in bringing our capabilities into harmony with the evolving socioeconomic changes which no one controls but all help to mold. Let us use those methods to cure our illness which we use daily in the cure of disease. If American Medicine must change, and change it must to survive, let us be the ones to control the change for our good and for the good of our patients. Change in itself is of little value, but a guided change in attitude, approach and effort is the only means whereby we may stop the present rapid decline of American Medicine.

J. J. L.

Some Notes and Cautions In Treatment of Stroke

Effective definitive treatment for cerebral infarction after cerebral thrombosis has occurred is not available. Moreover, we are unlikely to find agents or methods in the future which will do more than minimize the extent of brain destruction, and our therapeutic efforts in patients with completed stroke will continue to be directed toward return of maximal useful function in spite of continuing neurological defect. It is proper, therefore, to continue an aggressive search for preventive treatment and, in the same light, for those symptoms which warn of major catastrophe. The ultimate goal well may be a change in dietary habits of populations to prevent atherosclerotic disease, but this is of little immediate benefit to the patient in whom narrow hardened arteries have already developed. We will direct our attention, therefore, to the diagnosis and treatment of "stroke" *before it occurs* and ask if there are reasonably effective measures that may prevent this event.

Upon careful review, a large proportion (one half or more) of patients with cerebral infarction are found to have suffered from transient attacks of neurological dysfunction prior to their strokes. These transient focal ischemic attacks, often interpreted as "vasospasm," frequently may be related to reduced circulation in the distribution of major cerebral supplying vessels; the internal carotid and middle cerebral arteries on the one hand and the vertebral and basilar arteries on the other. Those symptoms formerly attributed to focal vascular spasm are more logically explained and demonstrated to be caused by atheromatous narrowing of major vessels, with blood flow passively dependent upon mean arterial pressure. These observations provided a first step of more than casual importance in preventive therapy. Many persons with vascular occlusive disease are found to have elevated blood pressure, and there is an understandable tendency for the physician to administer hypotensive drugs. Extreme caution is necessary. Hypertensive pressure levels may be required to force adequate blood for brain function through narrow vascular lumens. Reduction of blood pressure and flow may cause a major stroke, the very event we are trying to prevent. A first consideration is to maintain blood flow through narrow vessels and through tenuous collateral channels even by the use of pressor agents. Myocardial ischemia, paroxysmal abnormal rhythms and postural hypotension are among the more common events which may cause transient reduction in blood flow and result in episodic cerebral ischemia.

In recent years, much attention has been directed toward disease of proximal arteries in the genesis of stroke and of ischemic attacks. Many patients with these syndromes have been found to have narrowing or thrombosis of major vessels in the neck or even at the ostia at the aortic arch. Indeed, most patients with episodes of ischemia referred either to the carotid circulation or vertebral-basilar system are found to have large atheromatous plaques or complete occlusions in the major neck vessels. Attacks of hemiparesis, aphasia, or monocular blindness suggest disease in the carotid system. Attacks with such symptoms as diplopia, vertigo, alteration of consciousness, or bilateral visual phenomena suggest involvement in the posterior or vertebral-basilar circulation. Important diagnostic confirmation may be provided by the discovery of murmurs over the carotid bifurcation or the eyes with carotid atheromata, and over the subclavian arteries and behind the mastoids in vertebral artery narrowing. Retinal artery pressure may be significantly reduced ipsilateral to carotid occlusion and may be measured at bedside with a little practice. More conclusive diagnosis is made by arteriography in properly selected patients. Furthermore, by these methods, local atheromata in these vessels may be discovered at times in patients without (or before) symptoms. These plaques are often well localized, a fact which may be of considerable importance in treatment.

We have directed attention to one aspect of prevention, avoidance of blood pressure lower than the critical level for flow in narrowed vessels. Two additional approaches will be mentioned briefly; prolonged anticoagulation and surgical restoration of blood flow. Anticoagulant treatment has been found to be of little or no value in cases with completed stroke; indeed, it is probably

harmful and contraindicated. It may, however, be used with benefit in some patients with evolving strokes and in those with transient focal ischemic attacks. The initial enthusiasm for anticoagulants has now been tempered by time and experience. In random and controlled series of patients, proper prolonged anticoagulation does appear to reduce the frequency of focal attacks. There is, however, serious question as to the total effect. Not only may the anticoagulated patient have stroke, but the complication of serious or fatal hemorrhage is frequent enough to demand a high degree of caution. There is little doubt that devastating defect may be prevented or deferred in selected patients, but patients so treated must be carefully chosen.

Restoration of flow through a narrowed vessel to the brain by surgery has had increasing interest and attention. This is especially cogent because the narrowed segment or segments of artery may be restricted to vessels in the neck or thorax. Specific procedures include intimectomy or endarterectomy or bypass grafts and prostheses. It is too early to evaluate all of the results, but there is little doubt that flow can be restored through incompletely occluded arteries in many instances and that the patient may improve or stop having attacks. Few, if any, patients with total occlusions or completed strokes will benefit from surgery and the procedure should probably be reserved for those with transient attacks and incomplete atheromatous obstruction of flow. Both carotid and vertebral circulations should be visualized by x-ray arteriography prior to surgery. The former can be done in most instances by an experienced operator by percutaneous puncture of the common carotid artery in the neck. For the latter, retrograde filling from the subclavians may be required. In some, demonstration of the branches of the aortic arch may be necessary. Adequate visualization will frequently reveal disease at unexpected sites, thus altering the surgical approach or making it unwise. Surgery on vessels to the brain may be given serious consideration in patients who have symptoms related to atheromata in neck vessels, but probably should not be applied generally until long term effects have been evaluated. The procedure itself may precipitate stroke because of constriction of the vessel for performance of the surgery, embolization of atheromatous material, or institution of a propagated thrombus from the operative site.

Cerebral vascular disease will continue to be a serious problem, and its incidence will probably increase with an aging population. Continued investigations will undoubtedly bring more effective therapies and better methods of diagnosis *before the stroke*. Effective preventive treatment is available now for *some* patients; a number sufficiently great for the physician no longer to be satisfied with such incomplete diagnostic appellations as "little stroke" or "cerebral vasospasm." Each patient must be considered from these points: the extent and location of neural damage; the site or sites of vascular disease; the cause of the attack at the time it occurred and how recurrence may be prevented; finally, the treatment that is best for this patient, be it anticoagulants, surgery, or the simple withdrawal from hypotensive drugs.

Richard P. Schmidt, M.D.
Department of Medicine
Division of Neurology
University of Florida
College of Medicine
Gainesville

Eighth Annual Meeting Florida Diabetes Association Miami Beach, October 27-28, 1960

The Florida Diabetes Association will return to Miami Beach for its annual meeting late this month, and for the third successive year the Balmoral Hotel will be the headquarters. The dates are Thursday and Friday, October 27 and 28, and registration is scheduled for 9 a.m. on Thursday. Scientific sessions on Thursday morning and afternoon will be followed by a public meeting on Thursday night, sponsored by the Greater Miami Lay Diabetes Association. The scientific program will be continued on Friday morning and afternoon, with the annual business meeting scheduled for 1:30 p.m. on Friday.

Guest lecturers this year are Dr. Garfield G. Duncan, Professor of Medicine, University of Pennsylvania School of Medicine, Philadelphia; Dr. Alexander Marble, Physician, New England Deaconess Hospital, Assistant Clinical Professor of Medicine, Harvard Medical School, Boston; and Dr. Roger H. Unger, Assistant Professor of Internal Medicine, University of Texas Southwestern Medical School, Dallas. Member speakers on the program are Dr. A. Gorman Hills, Pro-

fessor of Medicine, Head of Division of Endocrinology, University of Miami School of Medicine, Miami; Dr. George F. Schmitt Jr., Assistant Clinical Professor of Pharmacology, University of Miami School of Medicine, Miami; and Dr. Joseph C. Shipp, Assistant Professor of Medicine, University of Florida College of Medicine, Gainesville.

Serving as moderators for the four scientific assemblies are Dr. Grover C. Collins, President, Palatka, Dr. William C. Thomas Jr., Gainesville, Dr. James B. Tobias, St. Petersburg, and Dr. Morris B. Seltzer, President-Elect, Daytona Beach. The program is approved for postgraduate study, Category I, by the American Academy of General Practice.

The fee for nonmembers is $25 and for members $15. Requests for reservations may be sent to Dr. George F. Schmitt, Secretary-Treasurer, 30 S. E. Eighth St., Miami.

EIGHTH ANNUAL MEETING
FLORIDA DIABETES ASSOCIATION
BALMORAL HOTEL, MIAMI BEACH, OCTOBER 27-28

THURSDAY, OCTOBER 27 — MODERATOR: Dr. Grover C. Collins

9:00	Registration	
10:00	"Prevention and Treatment of Complications of Long Term Diabetes"	Dr. Marble
10:45	"Acute Complications of Diabetes"	Dr. Duncan
11:15	Coffee Break	
11:30	"Hormonal Status of Endogenous Glucagon and Its Role in Blood Glucose Homeostasis"	Dr. Unger
12:00	Questions	
12:30	Luncheon Meeting — Board of Governors	

MODERATOR: Dr. William C. Thomas Jr.

2:00	"Elevated Cholesterol in the Diabetic: Significance and Approach"	Dr. Shipp
2:40	"The Detection of Hyperparathyroidism"	Dr. Hills
3:15	Intermission	
3:30	"Management of Diabetic Coma"	Dr. Duncan
4:10	Questions	
6:00	Cocktail Party	
8:00	Public Meeting sponsored by Greater Miami Lay Diabetes Association	

FRIDAY, OCTOBER 28

MODERATOR: Dr. James B. Tobias

10:00	"Overinsulinization, the Most Common Error in Insulin Therapy"	Dr. Unger
10:45	"Differential Diagnosis and Treatment of Chronic Hypoglycemia"	Dr. Marble
11:15	Coffee Break	
11:30	"Long Term Treatment of Patients With Diabinese" (Resident paper)	Dr. Edith J. Bradley
11:50	Questions	
1:30	Business Meeting	

MODERATOR: Dr. Morris B. Seltzer

2:00	"Diabetes Acidosis"	Dr. Schmitt
2:20	Intermission	
2:40	Panel Discussion: "Oral Hypoglycemic Agents"	
	"Use of Agents Other Than Tolbutamide"	Dr. Unger
	"Place of Oral Agents in Management of Diabetes"	Dr. Marble
	"Perspectives in the Treatment of Diabetes Mellitus"	Dr. Duncan
3:50	Questions	

Florida Academy of General Practice Meets in Jacksonville October 20-22, 1960

The Eleventh Annual Scientific Assembly of the Florida Academy of General Practice will be held on Thursday, Friday and Saturday, October 20, 21 and 22, at the Hotel Robert Meyer in Jacksonville. All Florida physicians are invited to attend, and there is no registration fee. The course is accepted for 10 hours Category I credit.

The program will be presented by eminent speakers from throughout the nation. They include:

Mr. Mac F. Cahal, Executive Director, American Academy of General Practice, Kansas City; Dr. Paul E. Craig, Member, American Board of Abdominal Surgery, Tulsa; Dr. Emerson Day, New York City; Dr. Temple Fay, former Professor and Head of the Departments of Neurosurgery and Neurology at Temple University School of Medicine, President of the Philadelphia Neurosurgical Society, Philadelphia; Dr. James G. Lyerly, Jacksonville; Dr. Peter F. Regan, Professor and Head of the Department of Psychiatry, University of Florida College of Medicine, Gainesville; Dr. Joseph R. Shaeffer, Director of Medical Education, Santa Rosa Hospital, San Antonio; Dr. Philip Thorek, Chicago; Dr. Leon Unger, Associate Professor of Medicine, Northwestern University Medical School, Chicago; and Dr. T. A. Watters, New Orleans.

Dr. Raymond R. Killinger of Jacksonville is general chairman of the Assembly Committee and also chairman of the Program Committee. The officers of the Academy are: President, Dr. Elmer B. Campbell Sr., St. Petersburg; President-Elect, Dr. James T. Cook Jr., Marianna; Vice President, Dr. Willard E. Manry Jr., Lake Wales; and Secretary-Treasurer, Dr. A. Mackenzie Manson, Jacksonville.

The program follows:

THURSDAY, OCTOBER 20

6:00 p.m. Dinner Meeting of the Board of Directors and Committee Chairmen

FRIDAY, OCTOBER 21

8:00 a.m. Registration. Exhibit Hall opens
9:30 a.m. Invocation, introductions and announcements
9:45 a.m. "The Listless Patient Syndrome," Paul E. Craig, M.D.
10:45 a.m. "Allergy of the Respiratory Tract—Diagnosis," Leon Unger, M.D.
Noon Buffet lunch in the Meeting Room
1:30 p.m. "Let the People Behold You," Mr. Mac F. Cahal
2:00 p.m. "The Use of Psychopharmacological Agents," Peter F. Regan, M.D.
3:00 p.m. "Allergy of the Respiratory Tract—Treatment," Leon Unger, M.D.
4:00 p.m. "Strokes, Subdural Hematoma and Brain Tumor," James G. Lyerly, M.D.
5:00 p.m. Visit Exhibit Hall
7:00 p.m. Social Hour
8:00 p.m. Dinner Party

SATURDAY, OCTOBER 22

8:00 a.m. Registration. Exhibit Hall opens
8:15 a.m. "The Cancer Detection Examination," Emerson Day, M.D. (film)
9:00 "Disaster Medicine, An Integral Part of the National Effort," Joseph R. Shaeffer, M.D.
10:00 a.m. "Basic Concepts in Teaching Non-Psychiatric Physicians Management of Psychiatric Problems," T. A. Watters, M.D.
11:00 a.m. "Practical Considerations in Diagnostic Screening of Cerebral Accidents and Chronic Types of Paralysis," Temple Fay, M.D.
Noon Lunch
1:00 p.m. Visit Exhibit Hall
1:30 p.m. "If I Had An Ulcer," Philip Thorek, M.D. (film)
2:00 p.m. General Business Meeting, Florida Academy of General Practice
3:00 p.m. "Cerebral Hydrodynamics and Important Recent Clinical Tests for Establishing Diagnosis in Neuromuscular Disorders," Temple Fay, M.D.
4:00 p.m. "Application of Psychiatric Principles in General Practice," T. A. Watters, M.D.

Southern Medical Association Meeting St. Louis, October 31-November 3, 1960

More than 6,000 physicians and guests are expected to attend the Fifty-Fourth Annual Meeting of the Southern Medical Association, to be held in St. Louis. The convention opens on Monday, October 31, and runs through Thursday, November 3. The association last met in St. Louis in 1954. This meeting will be its sixth in this "Crossroads of the Nation," which is well known for its hospitality as well as for its central location.

All meetings will be held in the conveniently situated and admirably appointed Kiel Auditorium, which will also house the several hundred scientific and technical exhibits. The host society is the St. Louis Medical Society, and the general chairman of the local committee on arrangements is Dr. Grayson Carroll. The president of the association is Dr. Edwin Hugh Lawson of New Orleans. Florida's councilor is Dr. Joseph S. Stewart of Miami, and Dr. Sullivan G. Bedell of Jacksonville serves on the editorial board.

More than 300 scientific papers are scheduled for the 20 sections. These programs are of unusual excellence and comprise a total of 46 half-day sessions, covering every phase of medicine. They will be presented by guest speakers nationally known in their special fields. In addition, a special feature this year will be two "first time" symposiums. On Monday morning, October 31, a Symposium on Cerebrovascular Disease will be a noteworthy attraction, and on Thursday morning, November 3, a Symposium on Medical Economics will no doubt evoke wide interest. Distinguished authorities have been selected for these two programs which offer points of interest to all physicians.

The president's guest, Dr. Dale Alford of Little Rock, Ark., will speak at the president's luncheon on Tuesday, November 1. Dr. Alford won his seat in the Congress as a write-in candidate and is up for re-election. At the president's dinner on Wednesday, November 2, presentation will be made of the Seale Harris Award for outstanding work in the field of internal medicine and the association's Distinguished Service Award.

Southeastern States Cancer Seminar for Physicians Orlando, November 16-18, 1960

The Tenth Biennial Southeastern States Cancer Seminar for Physicians will be held at the Cherry Plaza Hotel in Orlando on November 16 to 18. Highlighting the theme "New Horizons of Cancer Research and Therapy," an outstanding faculty of nationally prominent speakers will present a variety of subjects. The Seminar is being sponsored by the Orange County Medical Society in cooperation with the Florida Medical Association and is supported jointly by the Florida State Board of Health and the American Cancer Society, Florida Division, Inc. There is no registration fee. The course carries credit approval of the American Academy of General Practice.

The distinguished lecturers and their subjects are: Dr. Oscar Auerbach, Senior Medical Investigator, Veterans Administration Hospital, East Orange, N. J., "The Relationship of Smoking to Changes in the Tracheobronchial Tree and Lung Cancer;" Dr. Alexander Brunschwig, Professor of Clinical Surgery, Cornell University Medical College, "The Problem of Recurrent Cancer of the Cervix and Corpus Uteri;" Dr. Oscar Creech Jr., Chief of Surgery, Tulane University School of Medicine, "The Perfusion Technic for Cancer;" Dr. George Crile Jr., Head of Department of General Surgery, Cleveland Clinic, "The Possible Role of the Regional Lymph Nodes in the Body's Defense Against Cancer;" Dr. Joshua Edwards, Professor of Pathology, University of Florida College of Medicine; Dr. James Grace, Roswell Park Memorial Institute, "Viruses in Cancer;" Dr. John R. Heller, President, Sloan Kettering Institute, "Advances in the Chemotherapy of Cancer;" Dr. Lyndon E. Lee, Coordinator, Research in Surgery, Chief, Extra-VA Research Division; Dr. George E. Moore, Director, Roswell Park Memorial Institute, "The Spread of Tumor Cells and Their Importance to Chemotherapy;" Dr. Antolin Raventos, Associate Professor of Radiology, University of Pennsylvania School of Medicine, "New Methods in Radiation Therapy for Cancer;" Dr. Philip Rezek, Professor of Pathology, University of Miami School of Medicine; Dr. D. A. Ross, Chief, Physics, Oak Ridge Institute of Nuclear Studies, "Whole Body Counting Methods and Instruments;" Col. Frank M. Townsend, U.S.A.F., M.C., Director, Armed Forces Institute of Pathology, "The AFIP: Its Activities and Its Role in the Cancer Program;" Dr. Jerome A. Urban, Assistant Attending Surgeon, Breast Service, Memorial Hospital for Cancer and Allied Diseases, "Recent Trends and Treatment of Breast Cancer;" and Dr. Vincent Vermooten, Clinical Associate Professor of Urology, University of Texas Southwestern Medical School, Dallas.

Members of the Orange County Medical Society serving on the committee on arrangements for the meeting are Dr. Cecil G. Butt, Chairman, and Drs. Courtlandt D. Berry, James R. Cook, Harold W. Johnston, and Harold R. Torrance, all of Orlando. Dr. L. L. Parks of Jacksonville represents the Florida State Board of Health on this committee. For advance reservations or further information contact the 1960 Cancer Seminar Committee, 17 Lake Street, Orlando.

PHYSICIAN'S HOBBIES

Florida Physician-Artists Win Awards

The American Physicians Art Association held its Twenty-Third Annual Art Exhibit at the Miami Beach Auditorium and Exhibition Hall during the American Medical Association convention there June 13 to June 18, 1960. Florida was well represented with 49 entries from 20 physician-artists throughout the state. Out of 71 awards presented, Florida entries won 26 prizes in the various divisions which included Painting, Sculpture, Crafts and Photography, with Painting subdivided into Oil Scenic, Oil Still Life, Water Color, Portrait, Graphics and Abstract, and Photography subdivided into Color and Black and White. All categories were also divided into Beginner and Advanced Classes.

The 26 Florida awards consisted of four first prizes, five second prizes and three third prizes. The remainder were merit awards.

Dr. Ferdie Pacheco of Miami won a first prize for a Graphic in the Beginners Class entitled "Old Jazzman." Dr. I. Leo Fishbein of Miami Beach won a first prize for an Oil Scenic in the Beginners Class entitled "The Parthenon." Dr. Ben-Henry Rose of Miami Beach won a first prize for a Portrait in the Beginners Class entitled "Chinese Beggar," and Dr. Frank E. Bowser of Key West won a first prize for a Water Color, Advanced Class, entitled "Pennsylvania Farm in Spring."

Second prize winners included Drs. Wilfred Lansman and Jacob A. Glassman of Miami Beach, Ferdie Pacheco of Miami, Louis Lemberg of Coral Gables and Frank E. Bowser of Key West.

Third prize winners included Drs. David S. Howell and Ferdie Pacheco of Miami and Frank E. Bowser of Key West.

Honorable mentions went to Drs. Lawrence R. Medoff and Wilfred Lansman of Miami Beach and William Shedden of Ormond Beach.

All prizes were for paintings. Only three entries were made in the Craft category, but these did not win awards. There were no entries in the Sculpture or Photography divisions.

We are hoping for an even greater turnout from Florida for the Twenty-Fourth Annual Art Exhibit which will be in New York next June—more entries and more awards, particularly in Crafts, Sculpture and Photography. We are planning an art show for the Florida Medical Association convention in Miami Beach next May and expect to send it up to the American Physicians Art Association Exhibit in June. All those interested in exhibiting at the Florida Medical Association convention and/or the American Physicians Art Association Exhibit, in becoming a member of the American Physicians Art Association or in forming a Florida Physicians Art Association should contact me now at 1680 Meridian Avenue, Miami Beach.

Wilfred Lansman, M.D.
South Atlantic Regional Director
American Physicians Art Association

SHARE YOUR HOBBY

My hobby is _____

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Please reserve space for me to display it at the Annual Meeting in Miami Beach.

Signed _____

Mail to:
Thad Moseley, M.D., Chairman
Committee on Scientific Work
P. O. Box 2411, Jacksonville

OTHERS ARE SAYING

Democracy Is Not a Spectator Sport!

The Constitution of the United States says nothing about political parties. The Founding Fathers, in fact, looked upon parties as an evil, but a necessary evil. Yet political parties have become as vital to the proper functioning of democratic government as have the legislatures, the executive, and the courts.

The men who built this republic were proud to be politicians, because they were rugged individualists who had come to the new world to worship God in their own fashion and to run their own affairs. In their town meetings every man had his day and most spoke their mind boldly.

In Greece, the name for a town was "polis." Those who ran the town, its citizens, were "politikos." Our forefathers knew this and knew they had to be politikos, or politicians, to keep their freedom. They instituted government to secure our inalienable rights to life, liberty, and the pursuit of happiness. Government derived its power only from their consent. They proudly gave their time for it, for they wished to govern themselves.

We still have self-government and it still works, but it must have the *active* support of all the citizens. Over the years our country has grown and prospered and life is becoming more and more complex. People are no longer able to attend to all their needs themselves. Therefore we have to specialize. Our professional politicians are specialists too—specialists in making the laws or in administering them. The rest of the people are largely content with voting for or against them, and only about 60 per cent do that much.

In 1958 a total of 104,600,000 Americans were of voting age, but only 44,550,000 of them went to the polls to select their senators, congressmen, governors, state legislators, and so on. The senators and congressmen, governors, and others are the men and women who have been taxing us, spending our money, regulating us, telling us—in many cases—how we can run our businesses, resolving our place in the world. But a good deal less than half of us seem to care.

In 1956 some 102,000,000 Americans were old enough to vote, but only a little more than 62,000,000 were counted in the presidential contest, while 59,000,000 were all that cared about who made up the House of Representatives, and only 44,000,000 expressed themselves on the Senate.

An active, intelligent interest in government is an inherent part of good citizenship. After all, we were citizens before we received our degrees in medicine, and even after retiring from practice we will continue to be citizens and must play our role with vigor and courage.

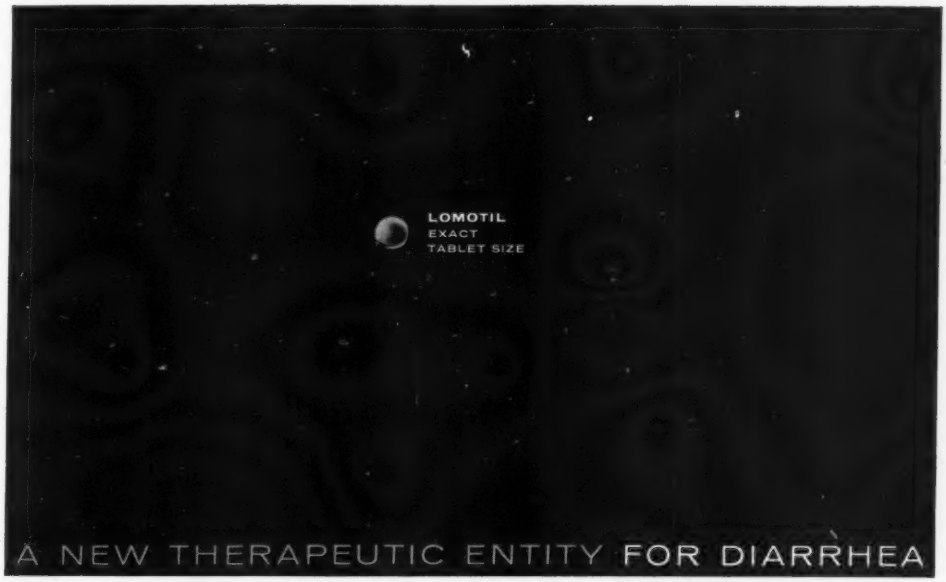
The first political obligation of each physician and his family is to register and vote in the party of his choice. Before he does register and vote, the physician should play an active role in the functioning of a party. As long as our country has a two-party system the candidates will be chosen and the platforms determined by those who participate actively in political affairs.

Most people confuse politics with issues. They do not know or they forget the "mechanics of politics," the mechanics of selecting, nominating, and electing citizens to public office. It means that politics and government should be in the mainstream of every adult's life. They are America. Freedom is something that is maybe the most glorious of all things. The entire complexion of life, liberty, and the pursuit of happiness changes abruptly when freedom passes by. Think of Hungary and some other countries. Think of many places where freedom does not exist.

History has demonstrated that governments begin to disintegrate when the people tend to depend on the "State" for everything. Paternalism is just as fatal as dictatorial power.

You, as an American, have an obligation to protect the independence and freedom which our forefathers gave to us. Nowhere else on earth at that time did people know the joys of being proud and free. It was considered the American experiment. News of it spread like lightning from shore to shore. This experiment is not dead. It has proved itself through years of stress and years of brilliance, from the town meeting to the halls of Congress. But are we as free today and will we be as free tomorrow?

The best way to preserve this freedom is to have millions of Americans taking an active part in government through politics. Responsible people from all walks of life should and must take an interest. Remember that our government can be no better than the men you elect. More and more of our citizens must get to know the candidates, know the issues, and take an active part in politics. Good government and freedom are



A NEW THERAPEUTIC ENTITY FOR DIARRHEA

LOMOTIL®

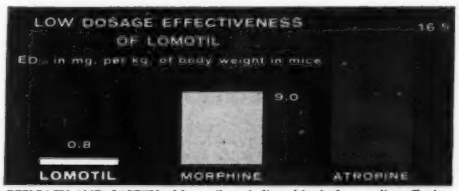
SELECTIVELY LOWERS PROPULSIVE MOTILITY

LOMOTIL represents a major advance over the opium derivatives in controlling the propulsive hypermotility occurring in diarrhea.

Precise quantitative pharmacologic studies demonstrate that Lomotil controls intestinal propulsion in approximately 1/11 the dosage of morphine and 1/20 the dosage of atropine and that therapeutic doses of Lomotil produce few or none of the diffuse untoward effects of these agents.

Clinical experience in 1,314 patients amply supports these findings. Even in such a severe test of antidiarrheal effectiveness as the colonic hyperactivity in patients with colectomy, Lomotil is effective in significantly slowing the fecal stream.

Whenever a paregoric-like action is indicated, Lomotil now offers positive antidiarrheal control ... with safety and greater convenience. In addition, as a nonrefillable prescription product, Lomotil offers the physician full control of his patients' medication.



EFFICACY AND SAFETY of Lomotil are indicated by its low median effective dose. As measured by inhibition of charcoal propulsion in mice, Lomotil was effective in about 1/11 the dosage of morphine hydrochloride and in about 1/20 the dosage of atropine sulfate.

PRECAUTION: While it is necessary to classify Lomotil as a narcotic, no instance of addiction has been encountered in patients taking therapeutic doses. The abuse liability of Lomotil is comparable with that of codeine. Patients have taken therapeutic doses of Lomotil daily for as long as 300 days without showing withdrawal symptoms, even when challenged with nalorphine.

Recommended dosages should not be exceeded.

DOSAGE: The recommended initial dosage for adults is two tablets (5 mg.) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is controlled. Maintenance dosage may be as low as two tablets daily. Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is supplied as unscored, uncoated white tablets of 2.5 mg., each containing 0.025 mg. (1/2400 gr.) of atropine sulfate to discourage deliberate overdosage.

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your responsibilities. If any group neglects to participate actively in the election of political leaders and the formation of political decisions, to that extent the democratic processes fail to function as intended by the authors of our Constitution.

The time has come for us to plunge into the cold waters of reality and to take an active part in political campaigns. What we are going to be able to do as medical men will depend upon *what* we are doing as *citizens*.

Learn the value of one vote—YOUR OWN. Democracy is not a spectator sport!

J. F. R.

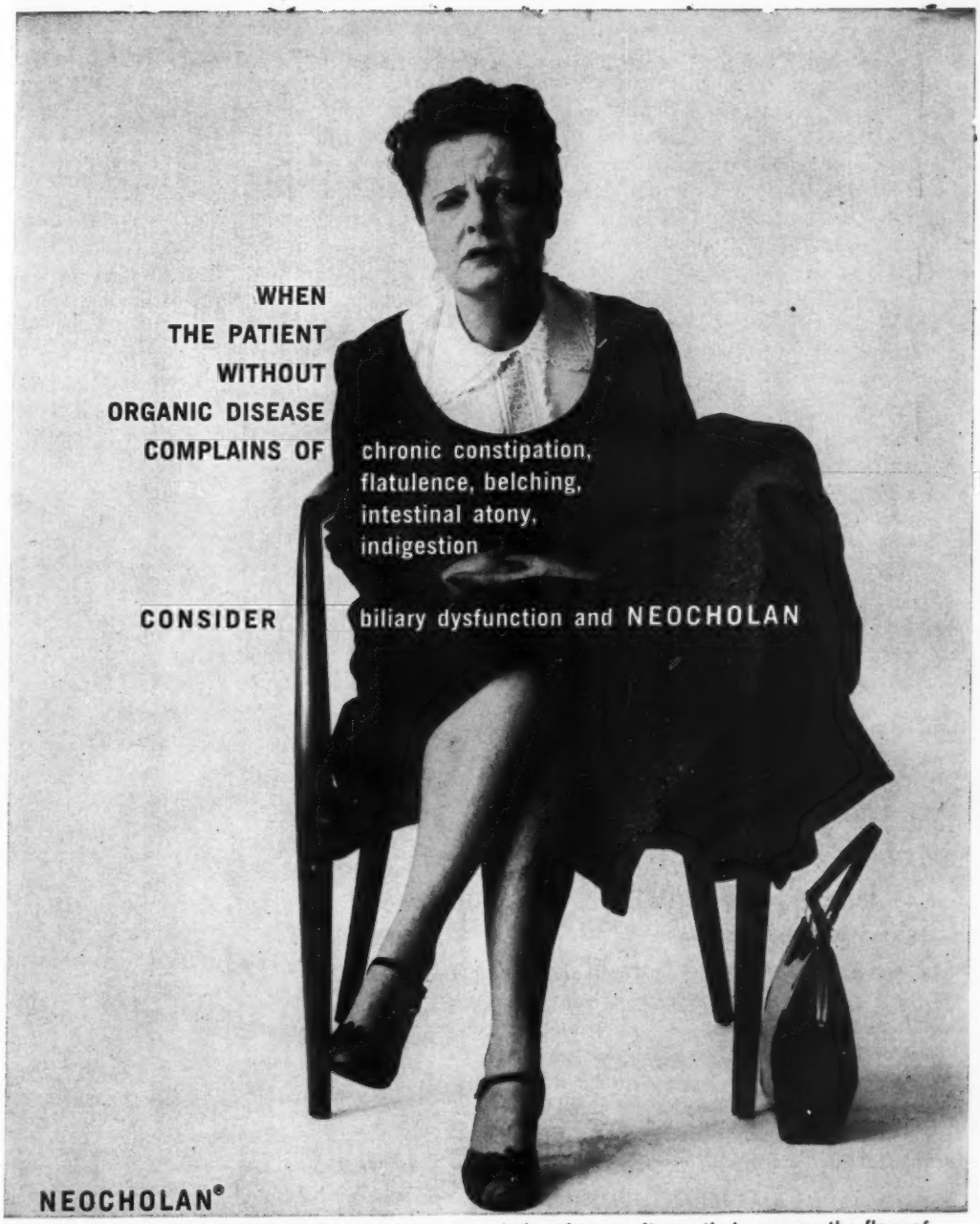
Reprinted from The New York State Journal of Medicine 60:2673-2674 (Sept. 1) 1960.

BLUE SHIELD

Advertising Carries the Message

PURPOSE.—Why have a Blue Shield? This has been asked—is continuing to be asked—by many persons, including some doctors. The answer, not lending itself readily to abbreviated explanation, can best be summed up by saying that so far it is the best method of prepayment yet devised against the expenses of illness, because it provides a professionally sponsored and supervised, voluntary plan with the greatest amount of coverage for the most people in all walks of life at the lowest cost. Included in this preferred protection are a high proportion of poor risks, with more than a fair share of the aged; made available through group coverage, conversion privileges without regard for age or health condition, and nongroup contract availability with no restrictions on age. Such practically unlimited coverage, from an underwriting standpoint, will continue to be available only if Blue Shield is strong.

STRENGTH.—In union there is strength. Trite truism of the times, it has never been more apropos than when applied to the partnership of the medical profession and Blue Shield. The strength of Blue Shield is that of the medical profession. Equally, the profession's position in opposition to the social planners has been strong and forceful because it has been able to point to Blue Shield as proof of the workability of the voluntary method. To remain strong and to utilize their strength to advantage, doctors must influence the thinking in their communities.



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Your patient will often respond promptly to Neocholan therapy. It greatly increases the flow of thin, nonviscid bile and corrects biliary stasis by flushing the biliary system. It also relaxes intestinal spasm, resulting in an unimpeded flow of bile and pancreatic juice into the small intestine. Neocholan helps to promote proper digestion and absorption of nutrients. It also encourages normal peristalsis by restoring intestinal tone.

Each tablet provides: Dehydrocholic Acid Compound, P-M Co. 265 mg. (Dehydrocholic Acid, 250 mg.); Homatropine methylbromide 1.2 mg.; Phenobarbital 8.0 mg. Supplied in bottles of 100 tablets.

PITMAN-MOORE COMPANY
DIVISION OF ALLIED LABORATORIES, INC.
INDIANAPOLIS, INDIANA

INFLUENCE.—The people of any community naturally look to their doctors for guidance in medical care and related fields. Each citizen is understandably concerned that some provision has been made to give him and his family protection against the unpredictable expenses of illness. Because of his high regard for his physician he also wants to make certain that his doctor is going to get paid when his services are required. Letting patients know that Blue Shield is the one program of prepayment approved and guided by their doctor will benefit patients, community and profession alike. Unequivocal support of the doctors' plan makes not only for strength, but progress in assuring perpetuation of voluntary methods.

PROGRESS.—It is with pardonable pride that physicians point to the remarkable advances in medical science in recent years. Progress in type and extent of prepayment protection has also been notable. When Americans want something, they usually find a way to get it. When their old problems remain unsolved by existing methods, then they seek new ways to cope with them. Whether the medical profession is happy with the situation is academic. Prepayment medical care is here to stay. Only through Blue Shield can there be assurance that it is properly guided and adequately controlled. Progress in prepayment will continue in the desired direction only if, as in medical care, the quality of service never becomes secondary.

QUALITY.—Just as there is no substitute for the doctor of medicine, there is no second best prepayment program for medical care. Aggressive support by the medical profession of the Plan which it originated, and over which it has control, convinces the public that doctors are capable of directing Blue Shield, and are thus less inclined to listen to suggestions that government can do better. Conversely, active support signified that plans without doctor guidance and control are inferior in the quality of protection provided.

During the last six months your Blue Shield has endeavored, through the advertising pages of this Journal, to remind Florida physicians that purpose, strength, influence, progress and quality are basic and indispensable to a prepayment plan acceptable equally to the public and to the medical profession. Following a principle of modern advertising technic you will find the number one advertisement, "Purpose," repeated on page 391

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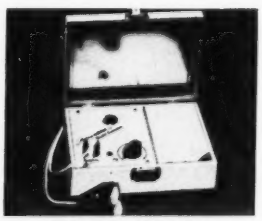

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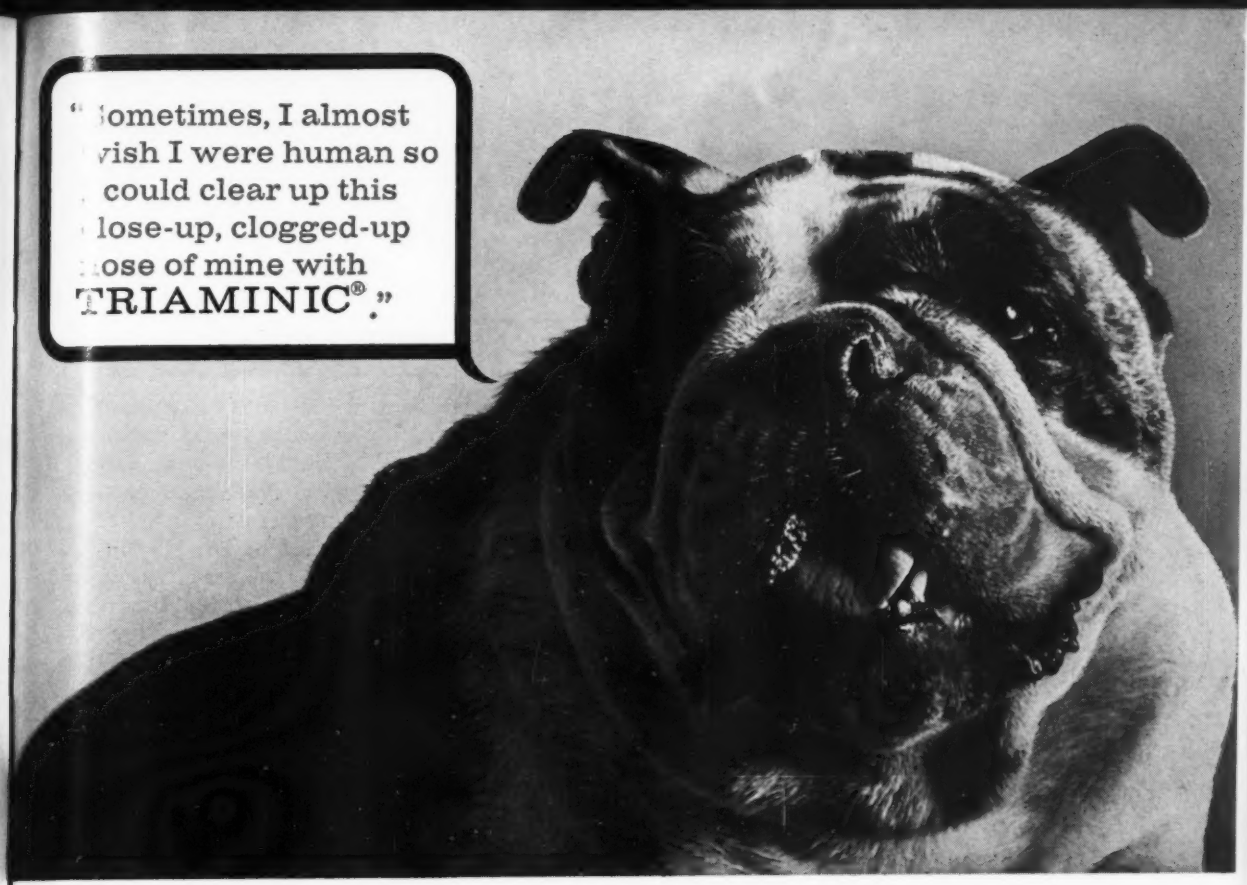
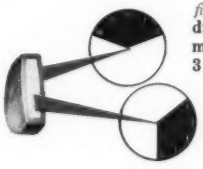


...and for humans with CLOGGED-UP NOSES...

Nasal congestion often persists with "bulldog tenacity." Nose drops and sprays often reach only the more superficial respiratory membranes and therefore fail to provide adequate relief. Furthermore, they may add to the patient's misery by producing rebound congestion, ciliary inhibition, and eventually "nose drop addiction." TRIAMINIC reaches *all* nasal and paranasal membranes *systemically* – provides more complete, longer-lasting relief while it avoids the harmful side effects associated with topical medication.

Indications: nasal and paranasal congestion, sinusitis, postnasal drip, upper respiratory allergy.

Relief is prompt and prolonged because of this special timed-release action:



first – the outer layer dissolves within minutes to produce 3 to 4 hours of relief

then – the core disintegrates to give 3 to 4 *more* hours of relief

Each Triaminic timed-release Tablet provides:

Phenylpropanolamine HCl.....50 mg.
Pheniramine maleate.....25 mg.
Pyrilamine maleate.....25 mg.

Dosage: 1 tablet in the morning, midafternoon and at bedtime. In postnasal drip, 1 tablet at bedtime is usually sufficient.

Each timed-release Triaminic Juvelet® provides:

½ the formulation of the Triaminic Tablet.

Dosage: 1 Juvelet in the morning, midafternoon and at bedtime.

Each tsp. (5 ml.) of Triaminic Syrup provides:

¼ the formulation of the Triaminic Tablet.

Dosage (to be administered every 3 or 4 hours):
Adults—1 or 2 tsp.; *Children 6 to 12*—1 tsp.;
Children 1 to 6—½ tsp.; *Children under 1*—¼ tsp.

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STOP *running noses* *and open stuffed noses orally*

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- d-amphetamine depresses appetite and elevates mood
- meprobamate eases tensions of dieting (yet without overstimulation, insomnia or barbiturate hangover).

Dosage: One tablet one-half to one hour before each meal.

A LOGICAL COMBINATION
IN
APPETITE CONTROL

Lederle

of this issue. It is significant that the plant world, which epitomizes all these fundamental characteristics is being used to supplement pictorially the general theme running through the messages.

STATE NEWS ITEMS

Dr. Hyman Sporn of Hollywood has returned from an extended trip abroad where he participated in two surgical conferences. From June 27 to July 7, Dr. Sporn was in Bordeaux, France, attending an otological functional microsurgery postgraduate course conducted by Dr. M. Portmann. From July 8 to July 31, he was in Vienna, Austria, where the conference on plastic surgery and microsurgery of the ear was conducted.

Dr. James N. Patterson of Tampa attended the meeting of the American Association of Blood Banks held in San Francisco on August 21-26.

Dr. Robert E. Zellner of Orlando was principal speaker at a recent meeting of the Orange County Pharmaceutical Association held in Orlando.

The Florida Society of Pathologists will meet in Sarasota on October 15-16, according to announcement by Dr. John A. Shively of Bradenton, secretary. Meeting jointly with the Society will be the Florida Division of the American Society of Medical Technologists. The societies will meet for the scientific session and dinner on the evening of October 15, but each will have separate business meetings.

A course for physicians in the latest developments in care, management and theory of the problems of prematurity has been scheduled for December 15-16 at Jackson Memorial Hospital, Miami. Credit will be allowed by the American Academy of General Practice. Apply to Bureau of Maternal and Child Health, Florida State Board of Health, P. O. Box 210, Jacksonville.

The annual meeting of the Southern Thoracic Surgical Association will be held November 17-20 in the British Colonial Hotel, Nassau, Bahamas. Information about the meeting may be obtained from Dr. Hawley H. Seiler of Tampa, who is secretary of the Association.

(Continued on page 436)

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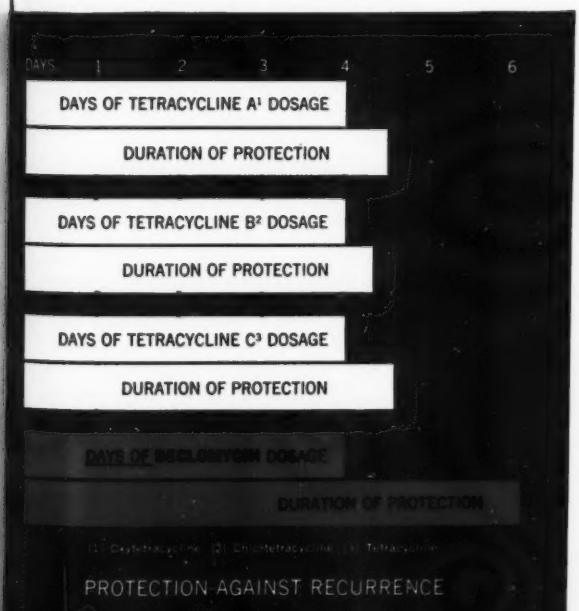
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DURATION OF PROTECTION
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SYRUP, 75 mg./5 cc. teaspoonful (cherry-flavored), bottles of 2 and 16 fl. oz. **Dosage:** 3 to 6 mg. per pound body weight per day—divided into 4 doses.

PRECAUTIONS—As with other antibiotics, DECLOMYCIN may occasionally give rise to glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis or dermatitis. A photodynamic reaction to sunlight has been observed in a few patients on DECLOMYCIN. Although reversible by discontinuing therapy, patients should avoid exposure to intense sunlight. If adverse reaction or idiosyncrasy occurs, discontinue medication.

Overgrowth of nonsusceptible organisms is a possibility with DECLOMYCIN, as with other antibiotics. The patient should be kept under constant observation.

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(Continued from page 432)

Dr. Joseph G. Seltzer of Orlando returned on September 20 from attending the Sixth International Congress of Internal Medicine held in Europe.

The Southeastern Allergy Association will hold its annual meeting on October 21-22 at the Atlanta Biltmore Hotel in Atlanta, Ga., according to announcement by Dr. Katharine B. MacInnis of Columbia, S. C., secretary-treasurer. Dr. Susan B. Dees, Duke Hospital, Durham, N. C., is in charge of the program.

The Twenty-Fifth Annual Convention of the American College of Gastroenterology will be held at the Bellevue-Stratford Hotel in Philadelphia on October 24-26. The program will consist of individual papers, a clinical pathological conference and motion picture films.

Dr. Louis L. Amato of Fort Lauderdale is among the 32 delegates designated by Governor LeRoy Collins for appointment to the White House Conference on Aging to be held in Washington January 9-12.

Dr. George W. Morse of Pensacola was guest speaker at a banquet held there recently which honored graduates of the School of Nursing of the Sacred Heart Hospital.

Drs. Francisco A. Hernandez and Milton S. Saslaw of Miami participated in the program of the Sixth Inter-American Congress of Cardiology held August 14-20 at Rio de Janiero. Dr. Hernandez took part in a panel on "Diagnosis of Congenital Heart Disease," and Dr. Saslaw presented a paper entitled "Prevention of Primary Attacks of Rheumatic Fever: Field Trial."

Dr. Richard M. Fleming of Miami presented an address on "Surgery and Surgeons for a Growing America" at a recent meeting of the Rotary Club of Miami Beach.

Dr. Spencer C. Manrodt of Melbourne was guest speaker at a recent meeting of District 31, Florida Nurses Association, held at Melbourne.

The Southeastern Region of the College of American Pathologists and the Virginia Society of Pathologists will hold a joint meeting at the John

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adult stable diabetes

"In our experience the action of DBI on the adult stable type of diabetes is impressive . . . **88% were well controlled by DBI.**"[1]

"Most mild diabetic patients were well controlled on a biguanide compound [DBI], and such control was occasionally superior to that of insulin. This was true regardless of age, duration of diabetes, or response to tolbutamide."[2]

"DBI has been able to replace insulin or other hypoglycemic agents with desirable regulation of the diabetes when it is used in conjunction with diet in the management of adult and otherwise stable diabetes."[3]

sulfonylurea failures

Among those diabetics who responded to tolbutamide initially and became secondary failures **DBI "gave a satisfactory response in 55%."**[4]

"DBI is capable of restoring control in a considerable portion of patients in whom sulfonylurea compounds have failed, either primarily or secondarily."[5]

"All twelve secondary tolbutamide failures have done well on DBI."[6]

"34 out of 59 sulfonylurea primary failures were successfully treated with DBI."[7]

Marshall Hotel in Richmond on November 25-26 on kidney disease.

The Department of Ophthalmology of the Emory University School of Medicine will sponsor a postgraduate course in ophthalmic surgery on December 1-2 in the auditorium of the Grady Memorial Hospital in Atlanta. Diagnostic principles and techniques, preoperative and postoperative management, and surgical principles and techniques in extraocular muscle surgery, cataract surgery and glaucoma surgery will be discussed by a distinguished faculty.

The Fall meeting of the Florida Orthopedic Society will be held at the Boca Raton Club, Boca Raton, on Saturday and Sunday, October 15-16. Members of the Society who are planning to attend should make arrangements directly with Mr. I. N. Parrish, Boca Raton Hotel and Club, Boca Raton, Fla.

An interim meeting of the Florida Urological Society has been scheduled for the weekend beginning Saturday, October 8, at Paradise Point, Crystal River, according to announcement by Dr. Henry C. Hardin Jr. of Miami, secretary-treasurer.

The Fall Meeting of the Florida Psychiatric Society is being held at Clearwater on October 15-16.

Dr. Joseph G. Matthews of Orlando was guest speaker at a recent meeting of the Lake County Medical Society held at Clermont. His subject was the complications of femoral fractures in children.

Dr. L. Roland Young of Daytona Beach was principal speaker at a recent meeting of the Daytona Junior Chamber of Commerce. The title of Dr. Young's address was "Fundamentals in Prevention of Nervous and Mental Diseases."

Dr. Henry F. Keiber of Winter Haven attended the meeting of the International College of Chest Physicians held in Vienna, Austria, August 28-September 2.

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(N1-β-phenethylbiguanide) is available as white, scored tablets of 25 mg. each, bottles of 100.
Send for brochure with complete dosage instructions for each class of diabetes, and other pertinent information.
1. Walker, R. S.: Brit. M. J. 2:405, 1959.
2. Odell, W. D., et al.: A.M.A. Arch. Int. Med. 102:520, 1958.
3. Pearlman, W.: Phenformin Symposium, Houston, Feb. 1959.
4. DeLawter, D. E., et al.: J.A.M.A. 171:1786 (Nov. 28) 1959.
5. McKendry, J. B., et al.: Canad. M. A. J. 80:773, 1959.
6. Miller, E. C.: Phenformin Symposium, Houston, Feb. 1959.
7. Krall, L. P.: Applied Therapeutics 2:137, 1960.
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Medical Licenses Granted

Dr. Homer L. Pearson Jr., secretary of the State Board of Medical Examiners, has reported that of the 401 applicants who took the examination of the Board, held June 20-21, 1960, in Miami Beach, 333 passed and have been issued licenses to practice medicine in Florida. The names of addresses of the 333 successful applicants follow:

Alfonso, Alvaro, West Palm Beach (National U. of Colombia 1949)
Alving, Ralph Eric, Miami (Columbia U. 1959)
Anderson, William Way, Miami (U. Louisville 1959)
Angelucci, Helen Marguerite, Fort Lauderdale (Woman's Med. Col. Pennsylvania 1927)
Armistead, James Davenport, Sebring (Emory U. 1960)
Armstrong, Louis Field, Plant City (U. of Miami 1960)
Bailey, William Ross Jr., Jacksonville (U. Florida 1960)
Baker, James Alva, Jacksonville (U. Miami 1960)
Baker, Marvin Israel, Jacksonville (U. Florida 1960)
Barancik, Maurice, Coral Gables (Northwestern 1960)
Barrow, Mark Velpeau, Sr., Crestview (U. Florida 1960)
Baumgarten, Harold, Miami (U. Miami 1960)
Baxter, Cecil Frederick, Gainesville (Harvard 1943)
Becker, Bruce Andrew, N. Babylon, N. Y. (U. Vermont 1955)
Beckmeyer, William Joseph, Fort Thomas, Ky. (Georgetown 1955)
Bennett, Etta Jean Lester, Gainesville, (U. Florida 1960)
Bernstein, Stuart Paul, Jacksonville (George Washington U. 1957)
Betcher, Albert Maxwell, New York (St. Louis U. 1935)
Blakey, Billy Ray, Orlando (U. Florida 1960)
Bleech, David Merle, Pahokee (U. Miami 1960)
Blick, Michael Samuel, Eggertsville, N. Y. (New York Med. Coll. 1937)
Blodgett, George Sumner, III, West Palm Beach (U. Miami 1960)
Bloom, Eugene Charles, Fort Lauderdale (U. Miami 1960)
Blum, Morton, Forest Hills, N. Y. (Albert Einstein Col. Med. 1960)
Boley, Santford Ripley, Miami (Wayne St. U. 1958)
Bong, Gary Michael, Miami (U. Miami 1960)
Bonner, Octavius Blanchard Jr., High Point, N. C. (Duke U. 1953)
Bono, Irvin David, Jacksonville, (U. of Miami 1960)
Bowen, Alfred Christian, Miami (U. Miami 1960)
Bradley, William Joseph III, Woodbury, Ga. (M.C. Ga. 1955)
Brame, Charles Lowel, Orlando (U. Miami 1960)
Branyon, Donald LeRoy Jr., Jacksonville (M.C. Ga. 1959)
Braunstein, Jonathan Jay, Miami (U. Miami 1960)
Brewer, Robert Allen, St. Petersburg (Indiana U. 1955)
Briant, Thomas Paul, Decatur, Ga. (Emory U. 1960)
Bridges, Barnard Tillman Jr., Birmingham, Ala. (N.Y.M.C. 1958)
Brown, Charles Seignious, Tampa (M.C.S. Carolina 1959)
Brown, Eli Hinson, Coral Gables (U. Miami 1960)
Brown, Warren Franklin, Atlanta, Ga. (U. Tenn. 1954)
Buttram, Thomas Latham, Fort Benning Ga. (Emory U. 1953)
Butts, James Allen, Atlanta, Ga. (Emory U. 1960)
Campbell, Selma Ronald, Fort Benning, Ga. (U. Miami 1960)
Carswell, Abel Paul Jr., Miami (Duke U. 1956)
Carter, Vernon Henry Jr., Little Rock, Ala. (U. Arkansas 1959)
Cary, Freeman Hamilton, Orlando (Emory U. 1950)
Chastain, Doyle Edward, Lake Wales (U. Miami 1960)
Cohen, Lawrence Samuel, Shaker Heights, Ohio (Vanderbilt U. 1960)
Cohen, Marshall, Richmond, Va. (M.C. Ga. 1958)
Cole, Edward Fowler, Orlando (Vanderbilt 1960)

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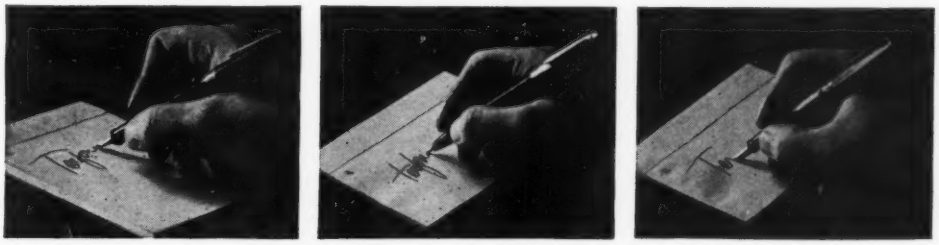

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Conlon, Harold Jay, Lakeland (U. Cincinnati 1945)
Constantine, Thomas Moore, Jacksonville (Duke U. 1954)
Crawford, Glenn Dale, Tampa (Emory U. 1960)
Cremata, Vincent Young Jr., Gainesville (U. Florida 1960)
Creston, James Ernest, W. Hollywood (U. Pittsburgh 1958)
Curry, Calvin Horace Jr., Quincy (Johns Hopkins 1960)
Daeppen, Charles Rudolf, Howell, Mich. (U. Zurich 1952)
Dain, Harvey Joshua, New York (U. Philippines 1940)
Dalton, Martin Lester Jr., Jackson, Miss. (M.C. Alabama 1957)
David, Arthur Kalil Jr., Jacksonville (Duke U. 1960)
Davis, Jack Alan, N. Miami Beach (Temple U. 1960)
Dillard, Richard Lynn, Springfield, Tenn. (U. Tenn. 1959)
Dockery, Junior Lee, Miami (U. Arkansas 1957)
Doenges, John Pell, Olney, Ill. (U. Maryland 1945)
Dowling, George Warren, Kermit, Tex. (Loyola U. 1952)
Drylie, David Marsh, Gainesville (Bowman Gray 1956)
Duke, James Roy Jr., Lakeland (Tulane 1960)
Eberly, Arthur Lee Jr., Lakeland (U. Miami 1960)
Eckels, Alan Ronald, Jacksonville (U. of Florida 1960)
Edelson, Stuart Roy, White Plains, N. Y. (U. Louisville 1959)
Ellis, Forrest Daryel, Homestead AFB (Indiana U. 1957)
Elstun, Wesley Bryan, Hammond, Ind. (Northwestern 1952)
Elton, Sanford Elsen, St. Petersburg (Northwestern 1959)
Engle, Mary Allen English, New York (Johns Hopkins 1945)
Entel, Irwin Leonard, Brooklyn (U. Geneva 1957)
Farmer, Charles Baring, Miami (U. Miami 1960)
Fedor, Richard Ernest, S. Miami (U. Miami 1959)
Ferber, Carl Jacob, Cleveland (Western Reserve U. 1946)
Ferrer, Meneses, Luis Manuel, Miami (Havana U. 1953)
Fischer, Robert Wallace, Miami (Duke U. 1960)
Fleites, Juan Arredondo, Fort Lauderdale (U. Havana 1948)
Foust, Betty Jean, Collingswood, N. J. (Indiana U. 1957)
Fox, Channing Henry, Miami (U. Miami 1960)
Free, Harry James, Gainesville (U. Florida 1960)
Friedman, Murry Nathan, Brooklyn (N.Y.U. 1943)
Gadboys, Howard Leonard, Coral Gables (Boston U. 1951)
Garlington, James Conway, Ocoee (Yale U. 1955)
Gersh, Herbert, Miami (U. Vermont 1955)
Gilchrest, James Warren, Greensburg, Pa. (U. of Pittsburgh 1944)
Gilman, Steve Hoyt, Tampa (U. Florida 1960)
Giordano, David Alfred, Durham, N. C. (Indiana U. 1955)
Golden, Howard, Miami (U. Miami 1960)
Goldstein, Arthur, Hollywood (U. Illinois 1960)
Goldstein, Burton Jack, Coral Gables (U. Maryland 1960)
Goldstein, Marvin Irving, Miami (U. Miami 1960)
Goodman, Stanley Sidney, Miami (St. U. N. Y. 1955)
Gordon, Meryl, Coral Gables (U. Miami 1960)
Grace, John Thomas, Marianna (U. Colo. 1958)
Gratz, Charles Jay, Miami Beach (Tulane U. 1960)
Griffin, William Vivian, III, Miami Beach (Emory U. 1960)
Gup, Alex Kramer Meyer, Pensacola (Emory U. 1960)
Gustafson, William Louis Jr., Miami (State U. N. Y. 1959)
Gutierrez, Gervasio Guzman, New York (U. Havana 1951)
Gutman, Elliott Michael, Miami (U. Miami 1960)
Hahn, Robert Emil, White Plains, N. Y. (Creighton U. 1958)
Hammer, Wray Allen, Fort Lauderdale (Emory U. 1960)
Hanley, John Patrick III, New Orleans (Tulane U. 1960)
Hanson, James Dewey, Miami Shores (U. Miami 1958)
Harris, Jeff Williams, New Orleans (Johns Hopkins 1952)
Harrison, Charles Elbert Jr., St. Petersburg (Emory U. 1960)

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Herrero, Brunildo Antonio, Miami (U. Florida 1960)
Herz, Ralph Jr., New York (Western Reserve 1953)
Hilbert, John Frederick, Atlanta, Ga. (Emory U. 1960)
Hillman, David Carl, Miami Beach, (U. Miami 1960)
Hiribarne, Pedro Fernando, Orlando (U. of Miami 1958)
Hogan, Walter Leroy Jr., Dallas, Tex. (M. C. Ala. 1957)
Holcomb, Allen Keith, Orlando (Emory U. 1960)
Holman, Franklin Pierce, Jacksonville (U. of Miami 1960)
Hoover, Walter Boyd Jr., Boynton Beach (Tufts U. 1953)
Hoppenfeld, Stanley, New York (Chicago Med. Sch. 1959)
Hunter, Richard Devois (Col.) Jacksonville (Meharry 1959)
Hurlburt, James Cole, Chapel Hill, N. C. (Duke U. 1958)
Hussey, Michael Ignatius, North Miami (Loyola U. 1957)
Jackson, Chrisman Spears Jr., Miami (U. Louisville 1959)
Jackson, Louise Oftedeal, Pompano Beach (George Washington U. 1949)
Jacobs, Robert Harry, Miami (U. Miami 1960)
James, Edward Milo, Savannah, Ga. (U. Miami 1960)
Jamieson, Robert Bruce Jr., Tangerine (Yale U. 1937)
Jarvis, Garth Louray, Galveston, Tex. (U. Michigan 1943)
Johnson, James Monroe, West Palm Beach (U. Miami 1960)
Johnson, Rand Carleton, St. Petersburg (Northwestern 1949)
Jonas, Adolph Garland Jr., Ponte Vedra Beach (Vanderbilt 1956)
Jones, Beverley Bryan, Miami (U. Va. 1954)
Jones, Billy Ernest, Daytona Beach (Duke U. 1958)
Jordan, Paul Howard Jr., Gainesville (U. Chicago 1944)
Kaminstein, Philip, Coral Gables (State U. N. Y. 1952)
Kann, Jules, Miami Beach (U. Basel 1959)
Kasnetz, Herbert Roy, Brooklyn (N. Y. M. C. 1960)
Katzen, Melvyn Joel, Miami Beach (U. Miami 1960)
Kempers, Roger Dyke, Lakeland (Wayne State U. 1954)
Kester, Richard Joseph, Jacksonville (Jefferson M.C. 1951)
Kiester, Kenneth Dwight Jr., Orlando (Oklahoma U. 1959)
King, Elmer Henry, Gainesville (Columbia U. 1955)
Koenig, Robert Louis, Mary Esther (Southwestern M. C. 1946)
Kohl, Stanley, Coral Gables (U. Zurich 1959)
Koon, Wiley Emmett, Lakeland (U. Miami 1960)
Kroll, Arnold Joseph, Miami Beach (Columbia 1960)
Kublin, John George, Detroit (U. Mich. 1960)
Kucera, Frank Edward, Delray Beach (U. Mich. 1955)
Kuhn, Beatrice Hart, Charleston, W. Va. (Duke U. 1943)
Lawrence, Duard, Miami (U. Louisville 1959)
Leatherman, James Wilson, Hot Springs, Ark. (U. Tenn. 1940)
Lemmert, William Arden, Tampa (U. Maryland 1956)
Lenton, Charles Trewartha Jr., Charlottesville, Va. (U. Penna. 1952)
Leonard, Samuel Anderson, New Orleans (Tulane U. 1960)
Lerner, George Gershon, Miami (Syracuse U. 1950)
Levine, Robert Allan, Cleveland Heights, Ohio (Howard U. 1959)
Lichtenstein, Robert Simeon, Fairfield, Conn. (Harvard U. 1952)
Liebman, Norman Charles, Miami (U. Penna. 1959)
Lighterman, Irwin, Miami (N. Y. U. 1958)
Lockhart, Herbert Garrison Jr., Linwood, N. J. (Temple U. 1955)
Logsdon, Donald Neil, West Palm Beach (U. Florida 1960)
Lutzner, Marvin Allen, Miami (Hahnemann 1955)
Luxenberg, Malcolm Neuwahl, Cincinatti (U. Miami 1960)
Lyons, Henry Richard, Washington, D. C. (Georgetown 1945)
Mahoney, William Dunvar, Venice (N. Y. M. C. Flower 5th Hosp. 1959)

(Continued on page 470)

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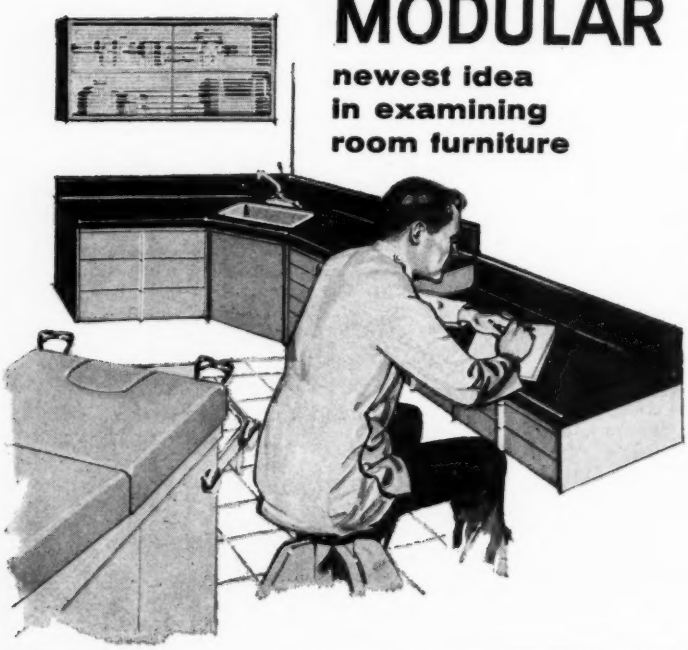

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Kestler, O.: Conservative Management of "Low Back Syndrome", J.A.M.A. 172: 2039 (April 30) 1960.

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(Continued from page 442)

Mangus, Jimmie Lee, Charleston, W. Va. (M. C. Va. 1959)
Mansell, John Lawson, Sanford (U. Penna. 1958)
Marcadis, Abe, Miami (U. Miami 1960)
Margolin, Frederick Ronald, Los Angeles (U. Miami 1960)
Margulies, Stanley Ira, Baltimore (Johns Hopkins 1960)
Markarian, Berge Harootium, Miami (U. Istanbul 1952)
Markman, Ira Eugene, New York (Chicago Med. Sch. 1959)
Mathews, Hurschell Frederick, Tampa (Bowman Gray 1960)
McConnell, James Franklin, Fort Lauderdale (Indiana U. 1959)
McCree, Douglas Gene, Oakland, Calif. (Emory U. 1957)
McLain, Arthur Lee, Tampa (U. Kansas 1957)
McLaughlin, Bernard Hugh, Eagle Lake (Temple U. 1954)
McLeod, Michael Eugene, Ft. Pierce (Duke U. 1960)
McMechan, David Robinson, Tampa (U. Mich., 1959)
McMurrain, Key David Jr., San Antonio, Tex. (Tulane U. 1958)
Mela, Henry Jr., St. Petersburg (U. Miami 1960)
Mendelblatt, Frank Irving, St. Petersburg (U. Miami 1960)
Menzel, Richard Edward, Miami (Georgetown 1957)
Merrell, Robert Alston Jr., New Haven, Conn. (Duke U. 1960)
Metzger, Charles Robert Jr., St. Petersburg (U. Florida 1960)
Miller, Gordon Roth, Miami (Washington U. 1960)
Mitchell, Fenton Martin, Norfolk, Va. (U. Pittsburgh 1957)
Mitchell, William Marvin, Baltimore (Vanderbilt 1960)
Mitchell, Williams Scales Jr., Orlando (Emory U. 1960)
Moench, George Cornelius, Treasure Island (U. Mich. 1958)
Moles, Stanley Sylvester, Gainesville (Duke U. 1959)
Muldrow, Louis Meriwether Jr., Jasper, Ala. (Vanderbilt 1960)
Muller, Walter Joseph III, Winter Park (Emory U. 1960)
Musinger, Jules, Miami Beach (N. Y. U. 1960)
Myers, Milton Bernard, Miami (U. Geneva 1956)
Nesmith, Marsh Arthur Jr., Plant City (Duke U. 1959)
Newman, Daniel Marquette, Fort Lauderdale (Indiana U. 1958)
Nicely, Donald Alvin, Monaca, Pa. (U. Pittsburgh 1957)
Nixon, William Anthony, Hialeah (U. Miami 1960)
Noah, Garrell Cela, Durham, N. C. (Emory U. 1959)
Nohr, David Albert, Perrine (U. Lausanne 1958)
Nolan, Raymond Paul, Pelham Manor, N. Y., (N. Y. U. 1951)
Nolan, Thomas Robert, Cleveland Heights, Ohio (St. Louis U. 1948)
Norenberg, Richard George, Tampa (U. Illinois 1959)
North, Luceil Bauer (f), Atlanta, Ga. (M. C. Ga. 1956)
Nystrom, Robert Bruce, Miami (Northwestern 1957)
Oberdorfer, Paul William, Jacksonville (Tulane U. 1960)
O'Brien, James Neil, Tampa (Marquette U. 1958)
O'Brien, John Stanley, Hartford, Conn. (U. Penna. 1959)
Ohlwiler, David Austin, St. Louis (U. Rochester 1954)
O'Lone, Joseph Patrick, Miami (U. Miami 1960)
O'Neal, Edwin Earl, Orlando (Tulane U. 1959)
Orgusaar, Rudolph, Woonsocket, S. D. (U. Erlangen 1949)
Orr, Louis McDonald Jr., Orlando (Emory U. 1960)
Ozaki, Charles Takeo, Gainesville (U. Florida 1960)
Paquelet, Charles John, Massilon, Ohio (Ohio State U. 1959)
Patterson, Joseph Richard, Tampa (Tulane U. 1958)
Patterson, Sam Polk, Memphis, Tenn. (U. Tenn. 1955)
Pearson, Clarence Arthur, Naples (U. Rochester 1960)
Pence, Robert Edward, Miami (U. Penna. 1954)
Pepus, Martin, Miami (U. Miami 1960)

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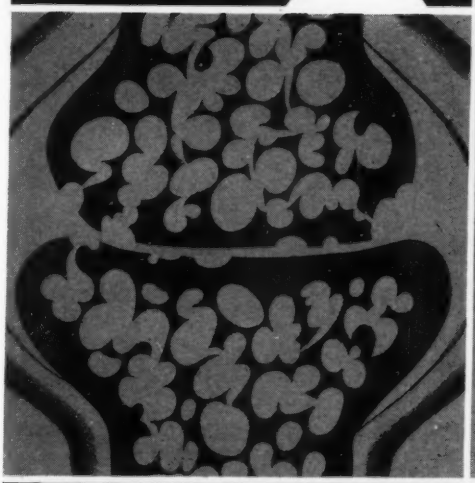

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Smith, Samuel Walker, Miami (U. Florida 1960)
Smock, Sidney Newberry, Ann Arbor, Mich. (U. Mich. 1960)
Snedeker, Bernard Carlton, Detroit (U. Mich. 1936)
Sobel, Arnold Meyer, Fort Campbell, Ky. (N. Y. U. 1955)
Sorosky, Arthur Dean, Los Angeles (U. Miami 1960)
Sotus, Peter Chris, Atlanta, Ga. (Emory U. 1956)
South, Edward Plumstead, Winter Haven (Hahnemann 1956)
Stahl, Norman Oscar, Brooklyn (Tulane U. 1958)
Stanford, Carol Carson, Jacksonville (Coll. Med. Evan. 1955)
Stanford, Thomas Albert, Jacksonville (Coll. Med. Evan. 1955)
Stephens, Hinson Lafayette, Jacksonville (U. Miami 1960)
Susong, William Anderson, Dallas, Tex. (U. Tenn. 1953)
Sweat, Arthur Warren, Largo (U. Florida 1960)
Tanenbaum, Mark Harris, Miami Beach (U. Miami 1960)
Taylor, Donald Willis, Ferndale, Mich. (Bowman Gray 1958)
Thorrens, Sheldon Jerome, Miami (Northwestern 1960)
Tobias, Donald A., Forest Hills North, N. Y. (Northwestern 1959)
Toker, Donald Leo, Miami (Western Reserve U. 1950)
Toledo, Florentino Humberto, Tampa (U. Havana 1953)
Traxler, Malcolm MacDougall, Gainesville (Emory U. 1960)
Trombino, James Frederick, Detroit (Hahnemann 1938)
Tufts, Thomas Wilson, Coral Gables (Ohio State U. 1959)
Turk, Lucas Newton III, Atlanta, Ga. (Emory U. 1952)
Turner, Leonidas Martin Jr., Tampa (U. Miami 1960)
Twente, Elmer William, East Syracuse, N. Y. (U. Kansas 1956)
Waldrop, Charles Wesley Jr., Miami (U. Louisville 1959)
Walton, Bruce Eugene, Gainesville (Western Reserve U. 1955)
Webb, Howard Warner, Decatur, Ga. (Emory U. 1957)
Weiner, Frieda Molly, Chicago (U. Illinois 1952)
Welch, George Harrison Jr., Durham, N. C. (Duke U. 1953)
Whalen, Charles Peter, Kankakee, Ill. (U. Illinois 1931)
Whiddon, Roscoe Owens, Tallahassee (U. Miami 1960)
Whitehurst, John Onan, Brooksville (U. Florida 1960)
Whittier, Millicent Kay Miller, Gainesville (U. Florida 1960)
Williams, John Webster Jr., Jacksonville (U. Miami 1960)
Williams, Robert Edwin, Homestead (George Washington U. 1956)
Wilson, John Thomas, Lake City (Albany M. C. 1954)
Wilson, Santford Russell Jr., Gainesville (U. Florida 1960)
Wirtanen, George William, Madison, Wis. (U. Florida 1960)
Wolfgram, Edwin Dale, Miami (State U. Iowa 1959)
Wood, Edward Howe, Lake Worth (U. Florida 1960)
Worton, Stanley Ivan, Miami Beach (Duke U. 1960)
York, Woody Neil, Tampa (Tulane U. 1958)
Young, William Joseph, Miami (U. Miami 1960)
Zatlin, Gabriel Stanley, St. Louis (Washington U. 1960)
Zeigler, Francis Marion Jr., Tallahassee (Emory U. 1960)
Zepernick, Richard Gustav, Pensacola (Tulane U. 1960)
Zimmon, David Samuel, Miami (Harvard 1958)
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BIRTHS AND DEATHS

Births

Dr. and Mrs. Joseph D. Foley of Jacksonville announce the birth of a daughter, Debbie Lynn, on July 5, 1960.

Deaths — Members

Copeland, Silas Morgan, Jacksonville	August 20, 1960
Colley, Sanford C., Tavares	June 29, 1960
Limbaugh, Louis, Jacksonville	August 1, 1960
Stansbury, Thomas C. Jr., Sarasota	May 3, 1960
Shaw, Wilfred McLaurin, Jacksonville	July 23, 1960

WOMAN'S AUXILIARY

Fall Conference and Board Meeting
Surf and Sand Hotel
Lido Beach, Sarasota
October 18-19, 1960

A cordial invitation to the Fall Conference is extended to all Auxiliary members who are interested. The local group is making plans that will insure FUN, FELLOWSHIP, AND FRIENDLY RELATIONS as well as INFORMATION AND INSPIRATION. The Conference opens with an informal coffee at 10 a.m. on Tuesday, October 18. Exhibits on all phases of Auxiliary work will be open at this time and Committee Chairmen will welcome your questions as well as your ideas and suggestions. I shall look forward to seeing you.

Mrs. John M. Butcher

Mama Due Real Vacation

At this time of year, it behooves us to give consideration to vacations for the female of the species. Frequently the mother and children take a "vacation" once a year or less. This possibly consists of packing, crowding into the car, driv-

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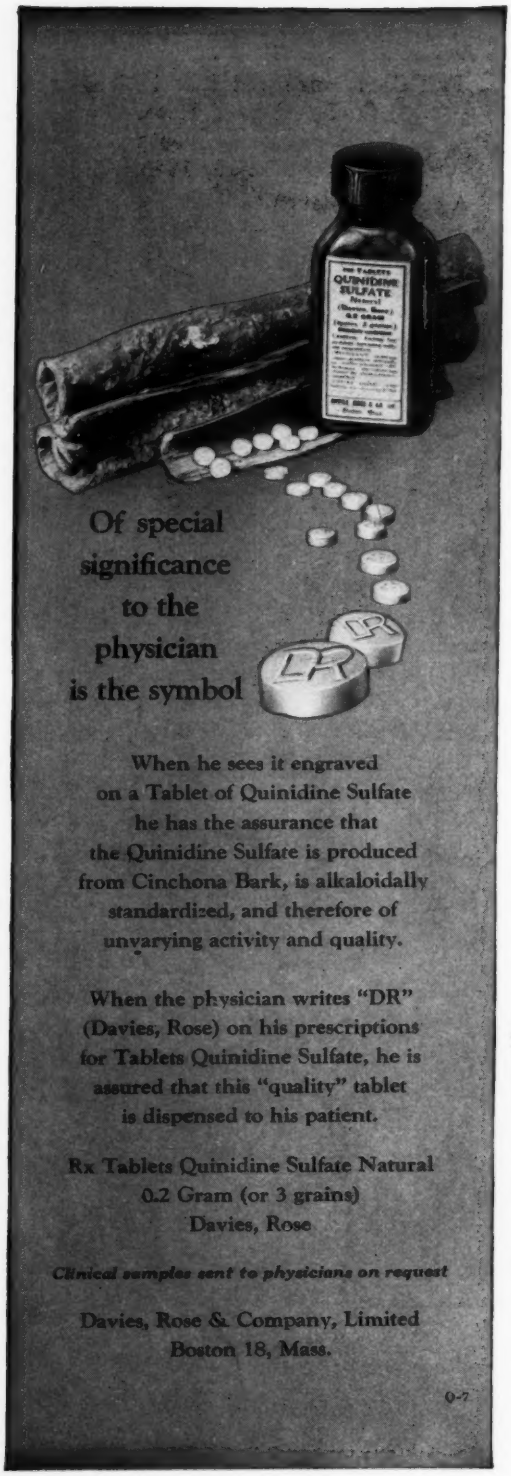
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ing like mad to the beach or the mountains, to live there in less than ideal housing and perform the same services that have been done for the rest of the year, plus the inconvenience of having inadequate facilities. This seems less than a real vacation for this tired mother ! !

It it amazing the number of women who never spend a night under a different roof from their child or children except for the times that they are in the hospital in the process of producing further children. It is my belief that it is utterly impossible to live on a higher mental plane than that of the youngest child if one is with that child or children continuously.

Years of "sameness" leaves little wonder that so many women are "tired mothers" until eventually they turn into "tired grandmothers." I feel strongly that adults need to live as adults with some degree of predictable regularity. It isn't necessary to get fancy and throw out of kilter a delicately balanced budget, but the prime requisite is to get away from the children and live as adults. This could be accomplished by even going to Kissimmee or Sanford and sitting in a rocking chair or going fishing. At least this would be different from the ordinary routine living day after day, week after week and year after year.

Of course, one must be satisfied that the children are taken care of adequately. There are some parents who have the unmitigated gall to assume that they are the only ones who can care for their children adequately, overlooking the fact that millions upon millions of children were reared very successfully by others. Self-reliance of the children would be enhanced if every waking breath weren't "mama," "mama," "mama," and becoming entirely dependent upon the same individual for everything. There is little question that a successful marriage would be greatly augmented if the husband and wife took these predictably regular times to live as two adults.

Robert L. Tolle, M.D.

NEW MEMBERS

The following doctors have joined the State Association through their respective county medical societies.

Active

Boling, Davis S., Tampa
Egolf, Robert L., Key West

(Continued on page 485)

(Continued from page 482)

Farrior, J. Lawrence, Pensacola
Glen, Robert S., Gainesville
Harrison, Paul W., Penney Farms
Lorincz, Andrew E., Gainesville
Malgrat, James D., Key West
Marshall, Mildred R., Gainesville
Reeves, John D. Jr., Gainesville
Ritch, Joseph G. Jr., Orange Park
Swanson, Eldon C., Green Cove Springs

Associate

Bass, Shelton T., Lakeland
Beck, Howard J., Miami
Brock, Charles L., Daytona Beach
Burman, Don M., Miami
Coury, Oswald H., Miami
Crews, Robert L., Coral Gables
LaRue, Roger P., Miami
McKey, Robert M. Jr., Miami
Molina, Vincent J. Jr., Miami

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Louis S. Goldstein Clinical Medicine 59:455 (1952) / Kass Archives of Internal Medicine Vol. 100, p. 709.

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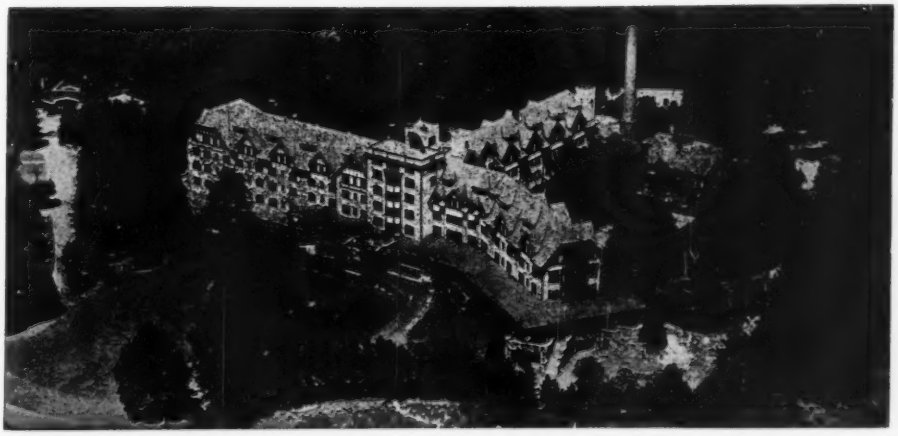

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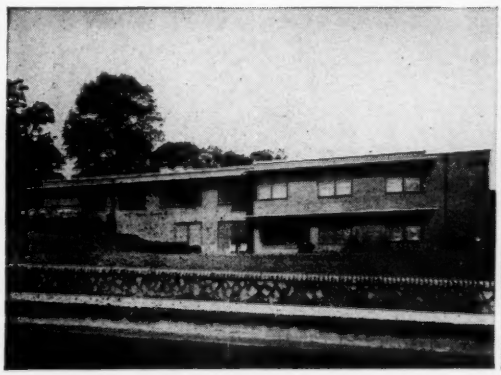

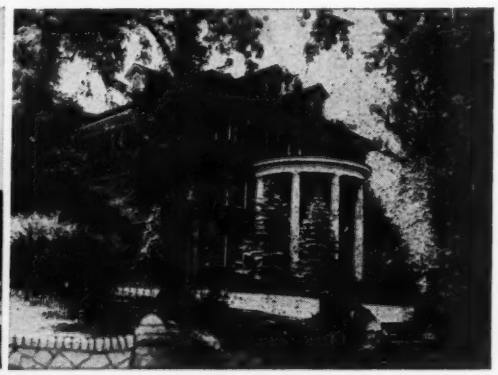

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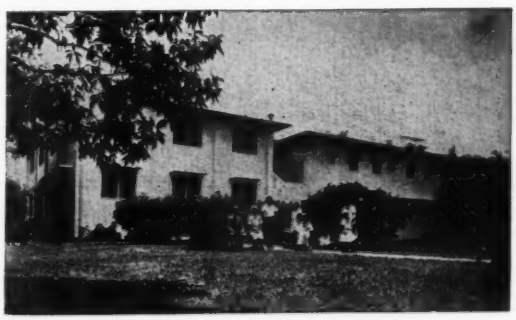
ORGANIZATION	PRESIDENT	SECRETARY	ANNUAL MEETING
Florida Medical Association	Leo M. Wachtel, Jacksonville	Samuel M. Day, Jacksonville	Miami Beach, May 25-28, '61
Florida Specialty Societies			
Academy of General Practice	Elmer B. Campbell Sr., St. Petersburg	A. MacKenzie Manson, Jacks'ville	Jacksonville, Oct. 21-22, '60
Allergy Society	I. Irving Weintraub, Gainesville	Ben A. Johnson Jr., Jacksonville	
Anesthesiologists, Soc. of	Richard S. Hodes, Tampa	J. Thomas Atkins, Jacksonville	
Chest. Phys. Am. Coll., Fla. Chap.	Ivan C. Schmidt., W. Palm Beach	Harold W. Johnston, Orlando	
Dermatology, Soc. of	Bruce M. Esplin, Miami	William C. Croom Jr., Jacksonville	
Health Officers' Society	J. Basil Hall, Tavares	James O. Bond, Jacksonville	
Industrial and Railway Surgeons	Fred H. Albee Jr., Daytona Beach	John H. Mitchell, Jacksonville	
Internal Medicine	William C. Blake, Tampa	Charles K. Donegan, St. Petersburg	
Neurosurgical Society	Irwin Perlmutter, Coral Gables	David H. Reynolds, Miami	
Ob. and Gynec. Society	T. Bert Fletcher Jr., Tallahassee	Sam W. Denham, Jacksonville	
Ophthal. & Otol., Soc. of	Kenneth S. Whitmer, Miami	Joseph W. Taylor Jr., Tampa	
Orthopedic Society	Michael DiCosola, Sarasota	Theodore Norley, W. Palm Beach	
Pathologists, Society of	John B. Miale, Miami	John A. Shively, Bradenton	Sarasota, Oct. 15-16, '60
Pediatric Society	Harry M. Edwards, Ocala	John H. Cordes Jr., St. Petersburg	Jamaica, Nov. 16-20, '60
Plastic & Reconstructive Surgery	Joseph E. O'Malley, Orlando	John M. Hamilton, St. Petersburg	
Proctologic Society	Don C. Robertson, Orlando	Matthew A. Larkin, Miami	
Psychiatric Society	Samuel G. Hibbs, Tampa	Merton L. Ekwall, Jacksonville	
Radiological Society	John S. Stewart, Ft. Myers	Alfred G. Levin, Miami	Ft. Lauderdale, Oct. 15-16, '60
Surgeons, Am. Coll., Fla. Chapter	Donald W. Smith, Miami	Charles Larsen Jr., Lakeland	
Surgeons, General, Fla. Assn.	Richard M. Fleming, Miami	Emmet F. Ferguson Jr., Jacks'ville	
Urological Society	H. Lawrence Smith, Tallahassee	Henry C. Hardin Jr., Miami	
Florida—			
Basic Science Exam. Board	P. A. Vestal, Winter Park	M. W. Emmel, Gainesville	Gainesville, Nov. 5, '60
Blood Banks, Association	Lloyd L. Newhouser, Miami	Faye Simington, Miami	
Blue Cross of Florida, Inc.	Mr. C. DeWitt Miller, Orlando	Mr. H. A. Schroder, Jacksonville	
Blue Shield of Florida, Inc.	Russell B. Carson, Ft. Lauderdale	John T. Stage, Jacksonville	Miami Beach, May 25-28, '61
Cancer Council	Joseph J. Zavertnik, Miami	Lorenzo L. Parks, Jacksonville	" " " " "
Diabetes Association			Miami, Oct. 27-28, '60
Dental Society, State	Wallace Mayo, Pensacola	Munroe Farber, Vero Beach	Miami Beach, May 21-24, '61
Heart Association	Gibson Hooten, Clearwater	Mrs. Alvin Savage, Miami Bch	Miami, May 27-28, '61
Hospital Association	Arthur L. Bailey, Orlando	J. F. Monahan Jr., Orlando	Miami, Nov. 30-Dec. 2, '60
Medical Examining Board	Robert T. Spicer, Miami	Homer L. Pearson Jr., Miami	Miami Beach, Nov. 20-22, '60
Nurses Association	Mrs. Idalyne Lawhon, Tampa	Mrs. Maurine Finney, Miami	St. Petersburg, Oct. 25-28, '60
Pharmaceutical Assn., State	L. W. Watson Jr., Marianna	Mr. R. Q. Richards, Fort Myers	W. Palm Beach, May '61
Public Health Association	Nathan J. Schneider, Jacks'ville	Everett H. Williams Jr., Jacks'ville	Miami Beach, Oct. 13-15, '60
Thoracic Society	George H. McCain, Tallahassee	Dwight J. Wharton, Jacksonville	Jacksonville, April 28-29, '61
Tuberculosis & Health Assn.	W. E. Arnold, Lakeland	Mrs. R. H. McIntosh, Port St. Joe	" " " "
Woman's Auxiliary	Mrs. John M. Butcher, Sarasota	Mrs. Richard V. Meaney, Palmetto	Miami Beach, May 25-28, '61
American Medical Association	E. Vincent Askey, Los Angeles	F. J. L. Blasingame, Chicago	New York City, 1961
A.M.A. Clinical Session			Wash., D.C., Nov. 28-Dec. 2, '60
Southern Medical Association	Edwin H. Lawson, New Orleans	Robert F. Butts, Birmingham, Ala.	St. Louis, Mo., Oct. 31-Nov. 3, '60
Georgia, Medical Assn. of	Milford B. Hatcher, Macon	John T. Mauldin, Atlanta	Atlanta, May 7-10, '61
S. E. Am. Urological Assn.	N. Lewis Bosworth, Lexington, Ky.	J. L. Campbell, Orlando	Hollywood, March 19-24, '61
Southeastern Surgical Congress	Walter C. Jones, Miami	A. H. Litton, Atlanta	Miami Beach, March 6-9, '61
S. E. States Cancer Seminar			Orlando, Nov. 16-18, '60
S. E. Hospital Conference	Gene Kidd, Nashville, Tenn.	G. C. Long Jr., Montgomery, Ala.	Memphis, April 19-21, '61
S. E. Allergy Association	David R. Thomas, Augusta, Ga.	Kath B. MacInnis, Columbia, S.C.	Atlanta, Oct. 21-22, '60



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County Medical Societies of Florida

SOCIETY	PRESIDENT	SECRETARY	MEETING DATE	TOTAL MEMBERS Associate	Active
Alachua	Eugene H. Cummings, Gainesville	Richard E. Perry, Gainesville	2nd Tues.		83
* *Bradford, Gilchrist, Union*					
Bay	Donald H. Anderson, Panama City	John L. Fishel, Panama City	2nd Tues.		33
Brevard	Cyrus E. Warden, Melbourne	Spencer C. Manrodt, Melbourne	1st Tues.	2	73
Broward	Anthony C. Galluccio, Hollywood	Frederick W. Fisher, Ft. Lauderdale	4th Tues.	37	267
Charlotte	Robert H. Shedd, Punta Gorda	Carl N. Reilly, Punta Gorda	2nd Tues.		7
Collier	Loral F. Gwaltney, Naples	Ethel H. Trygstad, Naples	4th Tues.		14
Columbia	Harry S. Howell, Lake City	Thomas H. Bates, Lake City	3rd Wed.		12
* *Baker*					
Dade	Franklin J. Evans, Coral Gables	DeWitt C. Daughtry, Miami	1st Tues.	124	1036
DeSoto-Hardee-Glades	Harold S. Agnew, Arcadia	Elmer J. Schmierer, Arcadia	1st Tues.		14
Duval	Floyd K. Hurt, Jacksonville	Albert D. Rood, Jacksonville	1st Tues.	5	357
Clay	Marcus B. Bergh, Orange Park	John M. Malone, Green Cove Springs			10
Escambia	William J. Overman, Warrington	Howard E. Herring Jr., Pensacola	2nd Tues.	1	120
Franklin-Gulf	Photis J. Nichols, Apalachicola	Joseph P. Hendrix, Port St. Joe	Last Wed.		7
Highlands	Hubert W. Coleman, Avon Park	Samuel A. King, Avon Park	3rd Mon.	1	13
Hillsborough	Linus W. Hewit, Tampa	Frank A. Massari, Tampa	1st Tues.	2	260
Indian River	Charles F. Rattray Jr., Vero Beach	Walter W. McCorkle, Vero Beach	2nd Tues.	2	14
Jackson-Calhoun	William R. Wandeck, Marianna	Francis M. Watson, Marianna	Quarterly		17
Lake	Arthur P. Buchanan, Leesburg	Thomas D. Weaver, Clermont	1st. Wed.	4	34
* *Sumter*					
Lee-Hendry	George D. Hopkins II, Ft. Myers	Leland K. Glenn, Ft. Myers	3rd Mon.		38
Leon-Gadsden-Liberty-Wakulla-Jefferson	Edson J. Andrews, Tallahassee	David J. McCulloch, Tallahassee	1st Mon.	8	76
Madison	Julian M. Durant, Madison	Thomas G. Bouland Jr., Madison	1st Tues.	1	6
Manatee	Willett E. Wentzel, Bradenton	John A. Shively, Bradenton	2nd Tues.	1	39
Marion	Herbert M. Webb Jr., Ocala	Keith H. Knorr, Ocala	3rd Tues.	1	33
* *Levy*					
Monroe	Walter R. McCook, Key West	Joseph L. G. Lester Jr., Key West	1st Thurs.		18
Nassau	David D. Bennett Jr., Callahan	Cecil B. Brewton, Fernandina Beach	1st Thurs.	2	10
Orange	W. Dean Steward, Orlando	Robert W. Curry, Orlando	3rd Wed.	29	231
* *Osceola*					
Palm Beach	Willard F. Ande, W. Palm Beach	Herman Baxt, W. Palm Beach	4th Mon.	26	191
Pasco-Hernando-Citrus	William H. Walters Jr., Lacoochee	W. Wardlaw Jones, Dade City	2nd Thurs.	1	21
Pinellas	Chas. L. Farrington, St. Petersburg	Whitman C. McConnell, St. Petersburg	1st Mon.	14	311
Polk	Charles Larsen Jr., Lakeland	Clarence L. Anderson, Lakeland	2nd Wed.	16	131
Putnam	Roy E. Campbell, Palatka	Norman B. Edgerton, Palatka	2nd Tues.		15
St. Johns	S. Raymond Cafaro, St. Augustine	Joseph A. Shelley, St. Augustine	3rd Tues.		17
St. Lucie-Okeechobee-Martin	Joseph H. Batsche, Fort Pierce	Maltby F. Watkins, Ft. Pierce	3rd Thurs.		30
Sarasota	James E. Kicklighter, Sarasota	Thomas G. Dickinson, Sarasota	2nd Tues.	5	89
Seminole	Edwin Epstein, Sanford	Robert J. Smith, Sanford	2nd Tues.	1	18
Suwannee-Hamilton-Lafayette	Frederick T. Mickler Jr., Jasper	Irby H. Black, Live Oak	1st Sat.		8
Taylor	John H. Parker Jr., Perry	John A. Dyal Jr., Perry	Last Fri.		5
* *Dixie*					
Volusia	Jas. J. Cunningham, New Smyrna Bch.	John J. Cheleden, Daytona Beach	2nd Tues.	6	100
* *Flagler*					
Walton-Okaloosa-Santa Rosa	Joseph C. Wilson, Ft. Walton Beach	Wm. W. Thompson, Ft. Walton Beach	3rd Tues.	4	33
Washington-Holmes	Walter H. Shehee, Chipley	L. H. Paul, Bonifay	Quarterly	3	4
				296	3,795
			Grand Total		4,091

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